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COMPREHENSIVE CARE IN THE TREATMENT OF CHRONIC SUPPURATIVE OTITIS MEDIA.*†

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The great scientific developments of the past few decades have made possible technical advances which have had a tremendous impact upon all fields of medical practice; including otology. Somewhat later in this period a broader concept of patient care, aimed at treating the "whole patient" and making use of all indicated medical and paramedical skills, has developed. While less dramatic than the technical developments, this already is having an influence upon therapeutic programs in our hospitals, an influence which, without doubt, will become increasingly felt as time goes on.

This is termed Comprehensive Care. It is not a discipline but an attitude. It is broader in its conception than Rehabilitation which is only a component of Comprehensive Care. It involves the concept of preventive medicine and of the influence, or effect, of socio-economic environment. It means going beyond the old objective of merely treating the disease. Obviously this is of greater importance in the management of chronic and recurring disease than in dealing with acute incidents, which most often may be managed satisfactorily by "target medicine." Naturally, Comprehensive Care seems

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particularly applicable to the treatment of chronic suppurative otitis media.

If one reviews the prevailing trends in the treatment of chronic suppurative otitis media during the past several decades, one cannot help but be impressed by a gradual changing pattern of management. Viewed at close-hand, it may seem divided into definite periods; from a distance, however, the pattern appears to have been a continuously revolving one. New scientific developments and mechanical improvements have revived procedures which had failed in the past but which now have been rendered possible of success. Oftentimes what now appears to be new, is in fact, the old, dressed up in new clothes, actually revolutionary in the sense of revolving. Sometimes this veneer of newness is so striking and so impressive that there is danger of ignoring the fundamentals which are time-proven and destined to endure.

In Politzer's textbook, which was the bible of otology in those earlier days, some 272 pages are devoted to the diagnosis and treatment of chronic suppurative otitis media. Much of this is perfectly valid today, and much seemed prophetic of what was to come. He described what he called the "extraction of the stapes," referring to the earlier work of Jack, and reported a case operated upon by his assistant, together with post-mortem findings. He stated his belief that the operation would be of practical importance in the future, in cases where the suppuration had been "exhausted." He described "intra-tympanic" operations to control suppuration but felt that ossiculectomy, while often temporarily successful, frequently would require mastoidectomy to effect a final cure. He stated that "intra-tympanic" operations to eliminate adhesive processes after cessation of the discharge, have a better chance for success than those operated upon for non-suppurative adhesions. He described Gromperz' operation in which small celluloid plates, 0.2 mm. in thickness, were introduced between the malleus and the promontory. All of these, and many more such procedures, were done without the aid of good illumination, the operating microscope, or the protection of modern antibiotic therapy. One is tempted to ask, "Is there

anything really new?" Isn't it just better methods for doing things, making use of modern scientific advances?

This was the era of Mastoid Surgery, of the radical and the modified radical procedures, and of the management of complications such as lateral sinus "thrombo-phlebitis," or of otitic brain abscess. It also was a period where great consideration was paid to nasopharyngeal and pharyngeal lymphoid tissue and to purulent sinusitis as common etiological factors. Careful attention was given to local treatment of the middle ear by which more, perhaps, was accomplished by meticulous cleansing than by the particular medicament employed. The "epi-tympanum" was irrigated by means of an attic canula. Granulations were carefully removed or cauterized by various chemical agents; and all of these measures together, the thorough removal of offending lymphoid tissue, meticulous mechanical cleaning; the control of granulation tissue, along with consideration of the patient's general health and well-being, often resulted in healing of the suppurative process and a fair degree of hearing function. This might be quite satisfactory, especially in unilateral cases. Radical surgery was reserved largely for cases with cholesteatoma, or with threatened complications.

Then came the era of chemotherapy, of antibiotic therapy, with a resulting decline in surgery; but the dream of a panacea was soon dissipated and mastoidectomy had its revival, and in new habiliments. Making use of good illumination and the operating microscope, surgical treatment for chronic suppurative otitis media became more definitive, with more emphasis upon preservation of hearing; but if one looks closely one sees much that was, at least, attempted by Politzer and his contemporaries. Apparently the cycle has completed its revolution. The radical operation of Stacke; the modified operation of Bondy, or of Heath; the paper patch of Blake; the stapedectomy of Jack; these were the prototypes for the modern radical and modified radical, the tympanoplasty and myringoplasty. These are all beautiful technical procedures; carefully selected and skillfully carried out with meticulous attention to detail, after thorough study of the "whole man," they can be construed as examples of Comprehensive Care.

The components of Comprehensive Care include Prevention, Diagnosis, Treatment and Rehabilitation.

Prevention depends upon early recognition, education of patients and the public, and correction or improvement of significant socio-economic factors.

Theoretically, and under ideal conditions, chronic suppurative otitis media should be preventable. Unfortunately, the otologist does not often have the opportunity of practicing prevention, at least directly. He may see relatively few cases of acute or recurrent otitis media and have little opportunity for management, including elimination of contributing factors. Pediatric myringotomies, blind reliance on antibiotics, unskillful adenoidectomies, or perhaps no treatment at all, provide grist for the surgical mill. Education of the general practitioner, pediatrist, the patient and his family, and the public would be helpful and should be a responsibility of otology. Incidentally, more emphasis upon the training of physicians permitted to perform tonsillectomies and adenoidectomies could prevent many cases of chronic suppurative otitis media.

Only indirectly can we correct and improve socio-economic conditions, by working for better Public Health agencies, such as medical social service and visiting nurse facilities. Correction of nutritional deficiencies, of poor sanitation, or hygiene; all these are important in prevention.

Diagnosis requires careful study and evaluation of the immediate disease, as well as of any contributing factors.

The diagnosis of chronic suppurative otitis media should be inclusive, encompassing every possible contributory factor and employing all indicated diagnostic studies and consultive skills. This cannot be done hurriedly and may require repetition. A well-taken history and a thorough examination, including careful functional tests, is essential and should determine the proper treatment. Overlooking significant nasopharyngeal lymphoid tissue, infected sinuses, allergic, or general debilitating conditions may prevent what might have been successful results of conservative measures. Shambaugh[1]

has pointed out that allergic states of the mucous membranes of the middle ear and Eustachian tube may predispose to recurring bacterial invasion, resulting in a stubborn although benign type of chronic suppurative otitis media. Cytological study of the secretions in the ear may reveal eosinophils and lead to the proper management. Sullivan,[2] also, has stressed the importance of recognizing and controlling allergy, particularly as a cause of acute exacerbation of a chronic process. This has been the experience of the writer. On many occasions control of the allergic process has resulted in a dry ear and with good serviceable hearing.

Treatment in a program of Comprehensive Care, may be surgical, medical, or both; as well as psychologic, psychiatric, or socio-economic. This may be local or general, or both; and may involve the use of many medical and para-medical skills.

Years ago Sir Felix Semon made a statement of far greater importance, and of greater validity, than his so-called Law: "Never let the magnitude of your procedure be greater than the severity of the symptoms you hope to relieve." This is not to decry surgical procedures when indicated but to suggest the broader viewpoint rather than the narrow focus on surgery alone. McNally[3] in his Presidential Address before the American Laryngological Association last year posed three similar questions in his summary.

"Is the condition serious enough to justify risk of total or partial destruction of the part under consideration?

"Is there no other possible or less dangerous procedure with prognosis nearly as good?

"Am I, the doctor, before God, prepared to assume the responsibility for the result as though I were the guardian of a minor patient, or as though that patient were my own child?"

Those of us who have practiced otology for an appreciable number of years could cite innumerable cases where persistence of meticulous local treatment, eradication or control of contributing factors and attention to general conditions have

resulted in cure of suppuration and with very serviceable hearing.

In reading the annual reports of certain hospitals I have been surprised to find in the list of surgical procedures, large numbers of radical mastoidectomies but no modified radical operations. One cannot help but wonder whether some of these would not have merited this more conservative procedure, the indications for which have been so adequately stated by Moore.[4]

Of equal and perhaps greater concern are those cases where radical operations of either type have been advised and carried out without careful study of all possible factors, or where an indicated and well-performed operation has been done but with little or no regard to other significant factors which might delay recovery, or tend toward recurrence of suppuration. The best of surgery is apt to prove disappointing if the patient has not been taught the necessity of proper after-care and how best to maintain himself in health.

Ferguson and MacPhail[5] in the preface of their book, "Hospital and Community," say: "Hospital treatment is usually only an episode in the general care of the patient and the health services cannot stand in isolation from other social services . . .

"There is a limit to what medicine can do to preserve fitness in the face of bad conditions of living and working . . .

"It is surely wasteful to spend large sums on expensive hospital care, only to return a man to living and working conditions that are almost certain to drive him back to the hospital in a few months."

Of course, such highly technical surgical procedures as tympanoplasty, for example, cannot be expected to be always 100 per cent successful. Proctor,[6] in one of the best articles on the subject, stated that tympanoplastic surgery is still in its formative stage. As such it obviously carries a calculated operative risk, but the many successful cases may warrant this risk; however, the fascination of this type of operation, at times, may be impossible to resist and results in its em-

ployment either at less skillful hands or when some more conservative measure would have been preferable. Wullstein[7] stated that the only contraindications to tympanoplasty were complications of the middle ear infection which were dangerous to life, and inadequate function of the internal ear. This might well lead to unnecessary surgery not picked up by the average Tissue Committee. It would seem better had he added the qualifying phrase, "when proven by adequate study as not amenable to more conservative management."

Schuknecht,[8] in the panel discussion on Tympanoplasty at last year's meeting of this Society, better stated the contraindications as "malfunctioning Eustachian tube, deficient middle ear mucosa, hyalinized collagen deposits in the middle ear, and age over 60"; and I am sure would agree to the above qualifying phrase.

Proctor's[6] standards for preoperative clinical investigation, stated as, "Careful otoscopic examination, complete audiometric assessment, sound probing and patch testing when feasible, tests for Eustachian tube patency, and radiological examination of the temporal bone," certainly qualify in a program of Comprehensive Care.

Comprehensive Care includes Rehabilitation: auditory, physical, or vocational, aimed at the preservation of all possible physical assets and the return of the patient to a useful and economically independent place in society.

In those cases in which treatment, medical or surgical, or both, has resulted in a cure of the infection with serviceable hearing, little else may be required. In those where the results have not been satisfactory and hearing is poor, auditory rehabilitation, by training in speech reading, and/or amplification by the use of hearing aids will be indicated. While a responsibility of the otologist, the services of the audiologist, the teacher of the deaf, the clinical psychologist, the social worker, and the vocational counselor may be required and be invaluable in providing rehabilitation. We have found the State Office of Vocational Rehabilitation particularly helpful in training and placing patients in employment. If evaluation

reveals any potential, if there be assets sufficient to suggest rehabilitation and a possible return to gainful employment, the services of this office have been readily available. This, in itself, is good medicine for the patient and does much to overcome the emotional frustration inherent in the handicapped.

Like all medicine, otology is revolutionary, constantly changing, as new scientific developments make possible improved methods and procedures, often seeming to resurrect techniques once discarded but now proven feasible. At the same time what is accepted today, may be discarded tomorrow; but through it all runs the search for the Truth and the constant urge to seek, find, and improve. It may take many skills to accomplish this, to solve the problems of even the one discussed here. Adopting the idea, the attitude of Comprehensive Care insured a broad approach without overemphasis on any one phase of therapy but consideration of the patient as a "whole man," not merely a discharging ear. It tends to keep things in their proper perspective.

SUMMARY.

Comprehensive Care involves the concept of preventive, curative, and rehabilitary medicine.

Prevention depends upon early recognition, adequate control of factors predisposing to infection or recurrence of infection, and upon education of patients, physicians, and the public.

Comprehensive Curative Medicine requires complete diagnosis, not only of the immediate otological condition but of the "whole patient," together with treatment aimed at control of all significant factors. Therapy should be as conservative as would be consistent with achieving the desired goal. Measures, whether Medical or Surgical, should meet the demands of the individual patient.

In order to achieve the optimum in convalescence, auditory rehabilitation as well as consideration of emotional or socio-economic factors, may be indicated.

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COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

October 23 to November 4, 1961.

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from October 23 through November 4, 1961, under the direction of Paul H. Holinger, M.D.

Registration will be limited to 15 physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

A NEW SYSTEM OF TYMPANOPLASTY USING VEIN GRAFT.*

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Rehabilitative surgery of the ear in chronic middle ear and mastoid disease has made great strides since the pioneering work of Wullstein and Zollner with tympanoplasty.[1,2] Technical difficulties, however, have limited the restoration of practical hearing to less than half of those with sufficient pathology to require procedures by-passing or substituting for portions of the ossicular chain. Chief among these difficulties are the problems arising from the use of a skin graft to cover the middle ear. In spite of the most careful attention to detail, the otologic surgeon must accept the possibility of flap cholesteatoma, dissolution of the graft with the recurrence of infection, formation of adhesions preventing free motion of the drum and ossicles, and the necessity of constant cleaning of debris from the surface of the graft; furthermore, the skin graft has excess mass and insufficient stiffness compared to the normal drum, causing poorer transmission of the higher frequencies.[3]

The use of vein as a grafting material was conceived by Shea and was first used in fenestration of the oval window as a covering for the oval window.[4] Later he extended this use to serve as a graft for small perforations and tears of the ear drum, both in stapes surgery and tympanoplasty.[5] Tabb has also reported on its use in myringoplasty.[6] Vein grafts have now proven to be vastly superior to skin as the sole grafting material for tympanoplasty.

The success of vein grafting is due chiefly to two factors:

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1. The vein is not used as a substitute for the missing squamous layer of drum, but as a replacement for the fibrous layer across which normal epithelium will grow. The graft is applied, therefore, to the inner surface of the drum remnant with the intima of the vein facing the middle ear. This layer eventually is covered by the cuboidal mucosa of the middle ear, but in the very early stages of healing it presents a smooth surface to the promontory and thus avoids adhesions. This also enables the surgeon to be sure how the graft is placed, a technical difficulty always troublesome with skin grafts.

2. The great hardiness of the vein as a free graft is the second major factor. Since the vein wall has very little intrinsic blood supply to recanalize, and lives mostly on tissue perfusion, lack of adequate nutrition is not a significant cause of failure in the early stages of drum remnant healing. The cutaneous epithelium of the ear canal and drum remnant grows quickly across the raw surface of the vein and brings abundant vascularity. The wall of the vein contains abundant smooth muscle and elastic tissue, and is even more resistant to the extrusive tendency of polyethylene implants than is the normal drum. We have also observed that vein has a superior ability to resist necrosis in the presence of infection in the middle ear, to that of a free skin graft.

The use of the vein has resulted in such a marked improvement in results over former techniques that it has been used exclusively during the past year in over three hundred tympanoplastic procedures.

TECHNIQUE.

The first step of the vein graft tympanoplasty is to remove and prepare the vein. This is taken routinely from the antecubital fossa. The larger of the lateral or median antecubital veins is chosen, and a 15 to 20 millimeter length is removed. The excess connective tissue surrounding the vessel is removed by dissecting off with a small blunt-pointed scissors. The vein then is stretched by opening the scissors within the lumen of the vein. After the vein is opened, it is laid aside until it is used in order to allow for any shrinkage

which might occur. It has been recent practice to place the vein back within the wound from which it was taken in order to keep it at normal body temperature and moisture. No vigorous stretching of the vein is done before use, as this can result in later contraction of the vein and subsequent small perforations occurring at the boundary of the graft and the drum.

The middle ear mucosa is anesthetized before anything is done, by placing gelfoam pledgets moistened with 4 per cent xylocaine hydrachloride through the perforation. The hairs of the ear canal are cut and the skin cleaned with ether followed by septisol.

The tympanoplasty is always performed as a transmeatal procedure. The middle ear and attic dissection is done through a large oval endaural speculum, held in place by a speculum holder permitting the free use of both hands. Ten to 16 power magnification is used throughout the operation. The first step in the procedure, as shown in Fig. 1, is to prepare the edges of the drum perforation to receive the graft. This is done before the exposure of the middle ear and attic in order to maintain enough tension in the drum remnant to strip the free edge of the perforation easily. Using a right-angle pick suction tip and very tiny cup forceps, the free edge of the drum is separated into two layers and its mucosal surface removed for a millimeter or two around the entire circumference of the perforation. If tympanosclerotic plaques are encountered in or on the drum, these are also dissected out and removed. It is frequently necessary to remove a squamous epithelial ingrowth from the promontory or other location in the middle ear or from the undersurface of the drum. Although the mucous membrane of the middle ear is preserved as much as possible, exposure of bare bone on the promontory does not increase the possibility of postoperative adhesions when closing the perforation with a vein graft.

In those cases with large perforations it has been found that the removal of pathology from the anterior two-thirds of the meso-hypotympanum is most easily done through the perforation, before incision and exposure of the attic space.

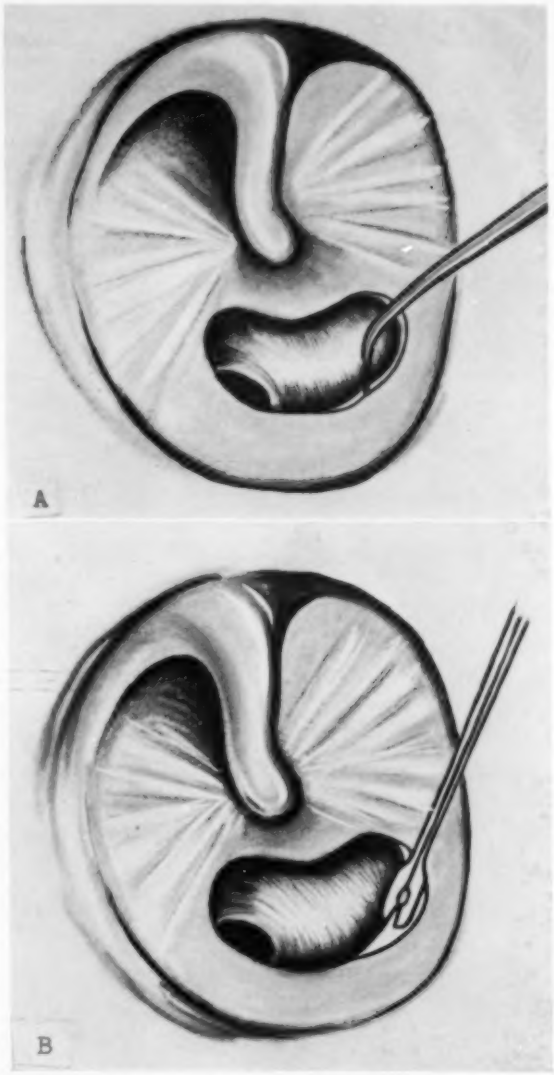

Fig. 1. Preparing the drum.
1a. The edge of the perforation is split into layers.
1b. The mucosa is removed from the underside of the drum.

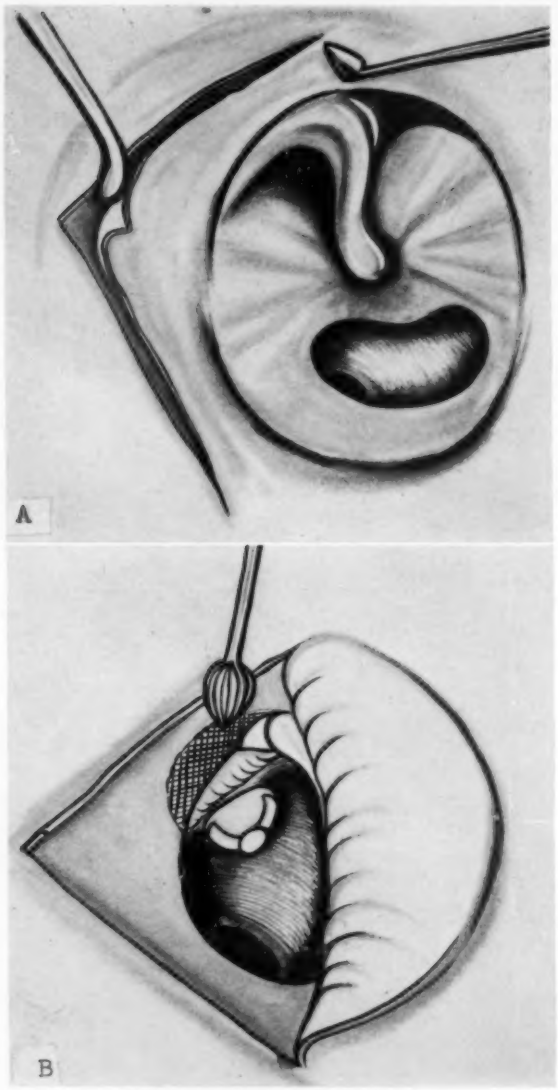

Fig. 2. Incision and exposure.
2a. Tympanoplasty incision.
2b. Amount of bone to be removed from attic wall for exposure.

Figure 2a shows the incision, which is a modified triangular stapes incision, beginning above the short process of the malleus about three or four millimeters out on the superior canal wall and reaching an apex posteriorly six to eight millimeters from the annulus. It is carried then to the posterior inferior portion of the canal ending three or four millimeters from the annulus. This insures a tympano-meatal flap large enough to obtain adequate exposure of the attic space, while retaining enough bony canal wall upon which to replace the flap. Exposure of the attic space is achieved by widening the notch of Rivinus and removing a portion of the posterior superior canal wall until a sufficient view is gained of the ossicular chain and attic pathology to permit dissection under direct vision. This may be removed widely enough to gain exposure of the aditus ad antrum. In doing this, care is taken to leave enough bony canal wall upon which to replace the tympano-meatal flap (see Fig. 2b).

The remainder of the dissection and reconstructive techniques depends upon the nature and extent of the pathology, so, of necessity, follows no set pattern.

OSSICULAR CHAIN.

The two most frequently encountered abnormalities of the ossicles are fixation due to fibrous or bony adhesion, and/or destruction of the long process of the incus.

In the case of an intact but immobile ossicular chain, it is often necessary to separate the incudo-stapedial joint to locate the point of fixation.

Stapes fixation may be due either to post-infectious bony or fibrous adhesions, tympanosclerosis, or otosclerosis. The arch of the stapes is amputated and the mucoperiosteum removed from the footplate. Since opening the oval window is unwise in chronic otitis, with or without a drum perforation, gentle footplate mobilization may be attempted at this time; but definitive fenestration of the oval window, if necessary, is always reserved as a second procedure following complete healing. The sound-conducting mechanism is reconstructed with a beveled polyethylene 90 strut extending

from the lenticular process of the incus to the footplate. In cases other than otosclerosis this is usually sufficient for lasting closure of the air-bone gap.

If the fixation occurs in the attic, mobilization of the head of the malleus and body of the incus is attempted first. If the malleus and incus can be freed, the incudo-stapedial joint is reapproximated. The chorda tympani then is cut at the posterior canal wall and wrapped around the reapproximated bones to secure a firm fibrous union. A common cause of fixation of the malleus is a bony stalk from the anterior part of the neck to the anterior rim of the attic.

In the case of firm fixation the procedure is the same as when the long process of the incus is gone. The incus is removed (see Fig. 3a). If necessary, the head of the malleus is amputated to secure free motion of the handle of the malleus. The arch of the stapes is removed by use of a circular saw attached to the small hand drill (see Fig. 3b). A polyethylene strut is then placed between the undersurface of the handle of the malleus and the footplate. The strut was first suggested by Shea for use in revising fenestration of the lateral semicircular canal failure cases. It is five and one-half millimeters long, notched on the end which fits under the middle of the handle of the malleus and beveled on the stapes end (see Fig. 3c). It has been found that the use of this strut has given much better hearing results than has myringostapediopexy or the use of a flat-headed columella directly to the undersurface of the drum. The columella must be used, however, if the handle of the malleus is absent. Fortunately, this is encountered only rarely.

CHOLESTEATOMA.

Cholesteatoma or squamous epithelial ingrowth in the attic and mastoid must be removed *in toto*. As the canal incision is made and the lateral attic wall taken down, attempt is made to dissect the matrix free from the walls of the attic intact, to be sure of its complete removal. This dissection is carried posteriorly as far as possible, but if the cholesteatoma extends into the mastoid antrum this is beyond reach of this

Fig. 3. Repair of sound conduction system.
3a. Removal of body of incus.
3b. Removing arch of stapes with crurotomy saw.
3c. Placing of malleolar-footplate strut.

exposure and it is necessary to enter the antrum directly for complete removal of the matrix. A separate endaural incision is made and a transcortical antrostomy is performed, maintaining the integrity of the posterior canal wall. This dissection is widened until the entire antral portion of the cholesteatoma may be seen and removed, in continuity with that already dissected in the attic space.

The endaural incision is then closed, leaving an enlarged antral space and the sound-conducting mechanism reconstructed as previously discussed. These steps are illustrated in Figs. 4a, b and c.

GRAFTING THE DRUM.

The elevated skin flap and drum remnant is now returned to its original position, taking care that the skin flap is in contact with the bony canal wall along the entire edge of the incision. The middle ear is packed with a suspension of finely divided gelfoam and a solution of polymyxin B powder and Ringer's solution, filling it to the level of the drum edge. This is prepared by mixing a 500,000 unit bottle of polymyxin B powder with 10 cc. of Ringer's solution to form a stock solution. At the time of use, ½ cc. of the solution is added to 2½ cc. of Ringer's solution, resulting in a final concentration of polymyxin B of slightly over 8,000 units per cc. A 1 x 3 cm. gelfoam pad is divided finely and added to the solution at the start of surgery, as in the method of Wullstein.

The vein is cut to approximate the shape of the perforation with a 2 mm. overlap around the entire circumference. If the perforation is heart-shaped, a notch is cut in the vein so that the edge of the graft may be approximated to the sides of the malleus rather than placing the vein underneath it. The graft is then carefully laid over the perforation with the intima inwards, being supported by the packing in the middle ear. By using a right angle pick, the overlapping portion of the graft is then tucked under the margin of the perforation and the squamous layer of drum carefully drawn out over the adventitial surface of the graft, so that there is no area where the squamous layer of the drum is turned

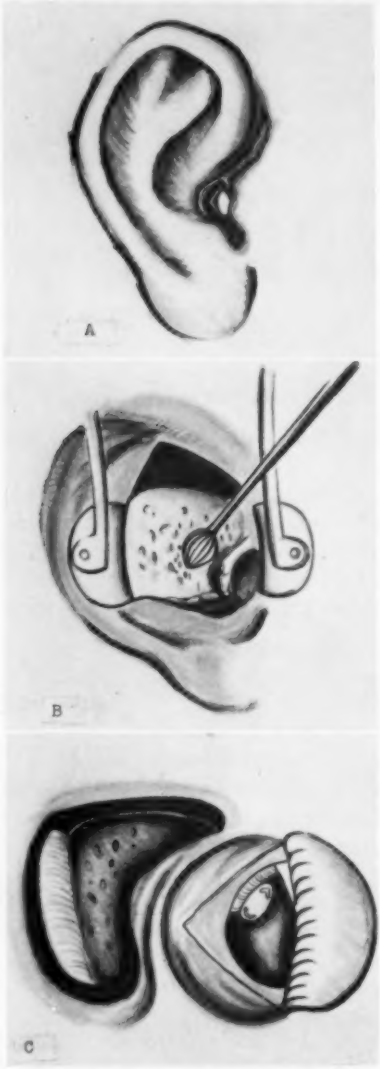

Fig. 4. Removal of mastoid pathology.
4a. Separate endaural incision is made.
4b. Transcortical antrostomy is performed.
4c. Antrostomy is enlarged sufficiently to remove pathology without sacrifice of posterior canal wall.

under (see Fig. 5a). This must be done under 16 × magnification since the edge of the drum has a tendency to curl inwards preventing good healing at that point. The graft and site of the canal incision also are covered with a small amount of the gelfoam packing and a cotton pad placed loosely in the outer ear canal (see Fig. 5b). Unless there is active infection present, no prophylactic antibiotic is given, since the infections that develop while using a prophylactic antibiotic are much more difficult to control. *No increased incidence of infection has occurred as a result of the non-use of prophylactic antibiotics.*

POSTOPERATIVE CARE.

The patient is discharged on the second postoperative day and is instructed to change the cotton tampon once or twice daily. He is cautioned not to sneeze with his mouth closed nor to inflate his own ears by blowing his nose. He is not to wash his hair or get water in his ear. A return appointment is given for three weeks later, since this has been found to be the optimum time for the first ear cleaning.

On the first postoperative visit, the ear canal is cleaned by suction, and the middle ear is inflated. Small areas of granulation on the drum may be touched with 10 per cent silver nitrate and gentian-violet. At this time the ear drum is usually found to be completely epithelized. The patient is given a 70 per cent isopropyl alcohol wipe to keep the ear clean and dry during the next few weeks. He is also instructed to inflate his ears by the Valsalva maneuver several times a day. Occasionally small perforations at the junction of the graft and drum occur. The edges of these perforations are cauterized with 50 per cent silver nitrate or trichloracetic acid, which usually results in a closure within a week or two.

RESULTS.

Over 300 procedures have been performed using this technique. Of these, 205 tympanoplasties and 20 myringoplasties have been followed for more than three months and are included in this report. One hundred cases have been followed

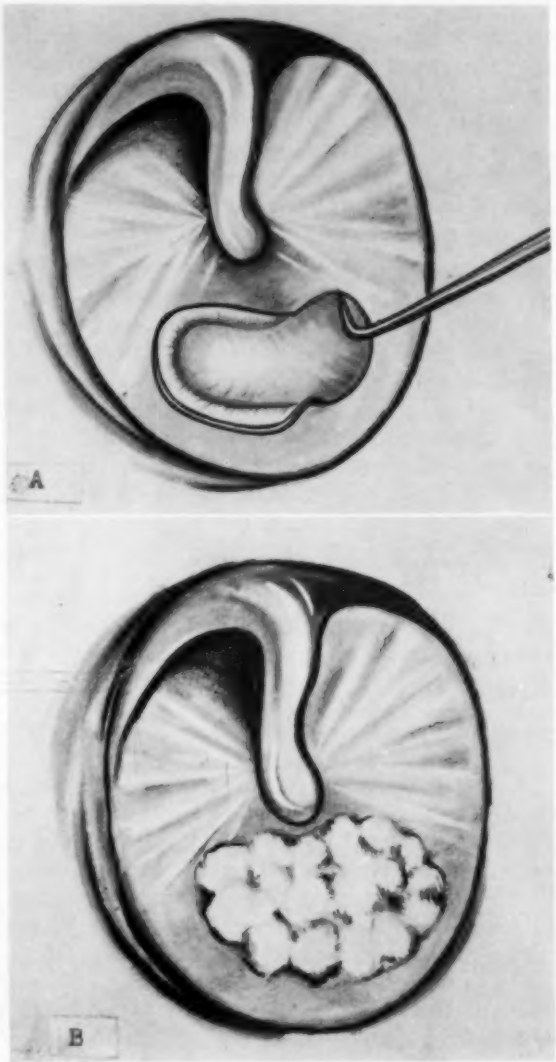

Fig. 5. Placing the graft.

5a. After filling middle with packing, vein graft is laid on the perforation and tucked under.

5b. Graft and incision are covered with small amount of packing.

for longer than six months and 125 cases have been followed for three to six months (see Table I). Of the 225 procedures performed, in 204, a vein graft was used. A primary take was achieved in 189, or 93 per cent. Of the 15 cases that did not achieve a primary take, this was evident at the first postoperative visit, 21 days after surgery, and not one primary take of the vein graft has broken down later over the period of observation.

The hearing results are summarized in Table II. A suc-

TABLE I.

Number of Cases	Myringoplasty	Tympanoplasty	Total
6-12 mos.	10	90	100
3-6 mos.	10	115	125
Vein Grafts	22	182	204
Vein Graft Failures	2	13	15

TABLE II.

Air-Bone Gap	Myringoplasty		Tympanoplasty		Total	
	6-12 mos. 10 cases	3-6 mos. 10 cases	6-12 mos. 85 cases	3-6 mos. 111 cases	6-12 mos. 100 cases	3-6 mos. 125 cases
Better than 15 db	10	9	77	69	87	78
15-30 db		1	7	27	7	28
Worse than 30 db			6	19	6	19
#Reaching 30 db level or closure of air-bone gap	10	10	78	78	88	88

cessful result is considered closing the air-bone gap within 15 decibels. The cases which have closed the gap to between 15 to 30 decibels have been sub-divided further to include those cases which have reached an air conduction level of 30 decibels or better. It may be noted that of the 100 cases followed for six months or longer, 87 cases, or 87 per cent closed the air-bone gap to within 15 decibels. Of the 125 cases followed from three to six months, 78, or 62 per cent closed the air-bone gap to within 15 decibels. Another ten achieved a hearing better than 30 decibels, resulting in a total success rate of 70 per cent. These two series taken together result in an over-all success rate for the entire group of 77 per cent.

During the course of the 225 operations, a total of 100 malleolar-footplate struts were used. In the analysis of these cases, it was found that 65 had closed the air-bone gap to within 15 decibels or less. An additional six cases had reached or exceeded the 30 decibel hearing level, giving a total successful result of 71 per cent. In comparison, myringostapediopexy was performed in 20 cases in which there was a sufficiently long stapes. A successful hearing result was obtained in 15, or 75 per cent. Although these results are quite similar, it was noted that in no case of myringostapediopexy was the air-bone gap completely eliminated. We feel this is due to the distortion of the drum created in attaching it to the stapes and to the difficulty of achieving a firm attachment to the stapes head. In the majority of the successful results with the malleolar-footplate strut, the air-bone gap was completely eliminated. In only two of the 100 cases did the strut later slip out of position.

COMPLICATIONS.

Most of the cases with an unsuccessful hearing result have been reoperated. The major cause of failure was the formation of scar around the prosthesis. This has occurred most often in those patients with poor tubal function, and those cases which organized rather than liquefied the gelfoam packing left in the middle ear. Reoperation served to improve the hearing in many of these cases.

Bone conduction remained constant or improved in all but one case. This patient had a severe episode of vertigo and vomiting on the second postoperative day, accompanied by complete loss of hearing. Exploration three weeks later revealed no obvious cause for the loss of cochlear function. This was the only case in which gelfoam was used to cover a small opening in the footplate.

This incidence of inner ear damage is of the same magnitude (0.5 per cent) as reported in other types of middle ear surgery.

COMMENT.

Although a much longer follow-up period will be necessary to assess the value of this method of tympanoplasty completely, it is apparent that the short term results with this technique have shown a striking improvement over skin graft tympanoplasty.

The great advantage of the use of vein has been that it allowed the precise reconstruction of the ossicular chain without extensive fibrosis or fixation. This has been demonstrated by the high degree of success in restoring useful hearing.

The procedure is so new that many details of the technique are, as yet, in transition. This is particularly true of the packing material used in the middle ear. The use of finely divided blood clot containing antibiotic as advocated by Buckingham[7] has been tried, and the initial results are quite encouraging. With this packing in the middle ear the grafts have been in a much more advanced state of healing at the time of the first postoperative visit, and there have been no cases of postoperative adhesion formation in this series.

NOMENCLATURE.

This approach to tympanoplasty varies somewhat in principle from that used formerly with the free skin graft. The repair of the ossicular chain and the repair of the ear drum, have been approached as separate problems. The nomenclature of Wullstein must be modified since his system is based on utilizing the graft to rebuild the sound conductive mechanism. Since the ear drum is repaired in the same way in all cases, the nomenclature applies only to the type of ossicular repair performed. The groups are as follows:

Type A. The ossicular chain is intact.

Type B. Portions of the ossicular chain are substituted while maintaining a near-normal sound conducting system, *i.e.*, polyethylene strut from the malleus to the footplate or from the incus to the footplate.

Type C. A polyethylene columella is used either from the

head of the stapes to the drum or from the stapes footplate to the drum. Myringostapediopexy also falls in this category.

Type D. A cavum minor is created with sound presented directly to the oval window. In no case has it been necessary to perform a type D tympanoplasty, the type C with artificial prosthesis being used in preference.

This technique has been used on all tympanoplasties during the past year. The size of vein obtained has been adequate for all perforations encountered, even those cases with complete absence of the ear drum. It is our opinion that vein is a much superior material with which to repair the drum, and return to the use of skin is not envisioned.

SUMMARY.

A new system of tympanoplasty with vein graft is presented. In 93 per cent of the cases in this series a primary take of the vein graft has resulted, and in no case has the graft broken down later. A successful hearing result was obtained in 77 per cent of the cases. A prosthesis constructed of polyethylene 90 tubing, extending from the handle of the malleus to the stapes footplate was described and the indications for its use given. This simplification of the technique has also a new system of nomenclature which is described.

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THE USE OF PARAFFIN TO CREATE A MIDDLE EAR SPACE IN MUSCULOPLASTY.*†

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In the search for a technique to provide hearing in deafness caused by chronic otitis, the main objective has been to find means for restoring a permanent air-containing middle ear cavity. Without a successful solution to this key step, no surgical technique can give a permanently satisfactory hearing result.

In ears in which there are major pathological defects in the mastoid as well as in the middle ear, the use of a system of skin grafting to re-establish a middle ear air space involves major difficulties. The primary aim in skin grafting must always be the preservation of the graft. This problem becomes a great deal more difficult when the center of the graft must bridge an air space which affords no blood supply. If drum remnants and ossicles, which may harbor infection, are retained in an effort to provide blood supply and support to the periphery of the graft, there is always the danger of burying sepsis or epithelium. If these structures are removed to allow for thorough exenteration of diseased tissue, an adequate blood supply for the graft is jeopardized; thus, the need to remove disease is always at odds with the need to preserve an adequate blood supply to the graft. This dilemma cannot be resolved so long as an uncertain system of skin grafting is used—that is, one which may fail because of immediate sloughing or delayed perforation of the graft as a result of poor blood supply, or return of disease due to inadequate removal of pathology.

As reported in previous papers,[1,2,3] the use of a temporal

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Boston, Mass., Jan. 6, 1961.

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muscle flap, with a self-sustaining blood supply through the pedicle, to bridge across the middle ear space, has eliminated these failures. Because the temporal muscle flap will remain permanently viable over an air space of any size, without need to garner blood supply from surrounding tissues, the surgeon has the great advantage of being able to remove all the diseased tissue with an initial wide-open radical mastoidectomy. The reliability of this approach is indicated by the fact that in 65 cases of musculoplasty performed so far, there have been no sloughs or delayed postoperative perforations in the reconstructed, substitute drums and no recurrence of disease in any of the ears operated.

Although the elimination of these failures has made musculoplasty a reliable procedure, one cause of failure remained for which no positive control was available. This was, in some cases, a collagenous tissue fill-in of the newly created tympanic space. It occurred where disease had destroyed the mucosa, or where the mucosa was diseased and had to be removed.

It has generally been recognized that the best insurance for maintaining a middle ear space postoperatively is to be able to leave a good mucosal covering over the promontory at the time of surgery; however, this is not always possible. The effects of cholesteatomatous or chronic adhesive disease, or polypoid and fibrous changes in the mucosa, often require complete removal of all tissue from the middle ear. When this is necessary, the denuded promontory faces the raw undersurface of the tissue covering the middle ear space, and fibrous tissue fill-in is almost a certainty.

In reporting a series of 26 cases of musculoplasty at the Workshop for Reconstructive Surgery in Chicago in March, 1959,[4] I pointed out that the only remaining cause for failure to reconstruct a permanent middle ear space in these operations was collagenous tissue fill-in in cases where an acceptable mucosa could not be left on the promontory. Since that time, in 39 consecutive cases of musculoplasty I have used a method which assures that a middle ear space will be maintained. This consists of filling the potential middle ear cavity

with paraffin before bridging this space with a temporal muscle pedicle flap. During the healing period between the first and second stages, if the mucosa has been removed from the middle ear, new mucosa grows from the mouth of the Eustachian tube around the paraffin prosthesis to line the potential middle ear space. In a second stage, the soft tissue covering the floor of the mastoid can be raised as a pedicle flap to expose the horizontal semicircular canal and the middle ear. The paraffin is then removed, the horizontal canal fenestrated, and the elevated flap replaced over the fenestra. In cases where the mucosa has been absent, I have not failed, so far, to see it regrow to form a mucosal-lined air space. In those cases where the mucosa has been acceptable and was not removed, the use of a paraffin prosthesis has been helpful in the proper placement and packing of the temporal muscle flap over the middle ear, as well as good insurance that the space will be maintained.

The purpose of this paper is to describe a controlled method for creating a permanent air-containing tympanic space, regardless of the type or amount of middle ear or mastoid disease present. The method has been developed and used as a step in the procedure of musculoplasty, an operation intended for use in those cases in which the ossicular chain has been destroyed, and the ear no longer has practical hearing. Musculoplasty is a standardized operation performed in two stages: the first stage consists of a radical mastoidectomy to eliminate all disease, plus use of a temporal muscle pedicle flap and a temporary paraffin prosthesis to reconstruct an air-containing middle ear space; the second stage, carried out after the first stage procedure has healed, consists of the use of the fenestration principle to re-establish a system of sound conduction to the inner ear. The technique is as follows:

OPERATIVE TECHNIQUE.

I. Musculoplasty, First Stage.

The scalp is shaved one inch around the auricle, and the ear and surrounding skin are prepared with ether, alcohol, and Zephiran chloride. The ear is draped with a transparent

plastic (Vi-Plastic*) in which a hole has been cut to accommodate the auricle. The edges of the plastic are glued to the surrounding skin (using Vi-Hesive*). Fluothane anesthesia is used. A 1 per cent lidocaine (Xylocaine) solution with 1:100,000 adrenalin is injected along the lines of the incision, to aid in the control of bleeding.

Endaural incisions are made, to develop anterior and posterior pedicle skin flaps from the outer half of the external auditory canal skin. The superior endaural incision begins half way in the canal and extends outward between the tragus and the crus helicis to the level of the crus inferior of the anthelix. That portion of the incision within the canal penetrates to bone, but care is taken to limit the outer incision to the depth of the temporal fascia. The inferior endaural incision begins about half way in on the floor of the external auditory canal and extends out of the canal at six o'clock. The origins of these two incisions are connected anteriorly and posteriorly by a circumferential incision. A posterior pedicle skin flap is then elevated from the bony canal with a large periosteal elevator and further elevated from the conchal cartilage by sharp dissection. A large semilunar piece of the exposed conchal cartilage with underlying soft tissue is removed to provide for a large endaural opening.

A self-retaining retractor can now replace hand retractors which have been used to this point. The skin on the posterior wall of the inner half of the external auditory canal is elevated and laid forward to expose the bony canal wall all the way to the notch of Rivinus. The mastoid is entered through the canal wall as advocated by Lempert.

(Alternately, if previous radical operations have been performed, the safest way to enter the mastoid is first to separate the upper part of the auricle from the mastoid bowl skin and retract widely. This may be done after making a superior endaural incision which extends upward and posteriorly over the auricle. A line of cleavage is obtained between the auricle and the temporal fascia and the upper part of the auricle is separated from the temporal muscle and turned downward.

*Aeroplast Corp., Dayton, Ohio.

A further circumferential incision is made beneath the auricle at the level of the cortex to separate the skin lining the superior and posterior part of the mastoid bowl from the overlying soft structures. The auricle is then retracted with a large periosteal elevator until the bony rim posterior to the mastoid bowl is exposed. With complete exposure of the mastoid cavity, the lining skin, or diseased soft tissue, may be elevated more safely. If there are exposures of dura or sinus, these can be uncovered with better vision, and in the event of accidental injury to the sinus there is complete access to the area.)

A thorough and meticulous wide-open radical mastoidectomy is done, exploring the mastoid to its anatomical limits and removing all diseased tissue. After removal of the bony posterior canal wall the superior spine is completely taken away and the inferior spine or facial ridge is reduced to a maximum. The floor of the external auditory canal is lowered to the level of the hypotympanum. The middle ear is then inspected. If there is healthy mucosa covering the promontory it is not disturbed. If the mucosa is diseased or roughened, or if there is cholesteatomatous matrix or squamous epithelium present, all the soft tissue of the middle ear is removed to the mouth of the Eustachian tube. Where there are granulations or infected cells in the hypotympanum or over the carotid plate, these are thoroughly removed. The mouth of the Eustachian tube is never curetted. A bougie is sometimes passed from the middle ear to the pharynx to establish patency of the tube.

The skin covering the outer half of the anterior wall of the external auditory canal is now elevated to the external meatus to form a pedicled skin flap. This requires sharp dissection and is best accomplished with a pair of fine curved manicure scissors, care being taken not to cut the underlying cartilage.

The temporal muscle is next exposed by extending the superior endaural incision upward and posteriorly over the auricle and by separating the loosely attached scalp from the temporal fascia. With hand retractors the scalp is tented to

give access to the posterior limits of the temporal muscle. A flap is cut from the temporal muscle with its pedicle anterior. The flap is taken from the lower edge of the muscle and measures about ½ inch at its pedicle, fanning out in a club-shaped fashion to about one inch in diameter at its posterior end. The flap should be made as long as possible, extending to the limits of the temporal muscle or even beyond. If a flap is not cut which appears longer than necessary, it may prove too short to reach and cover the middle ear comfortably because of surgical edema and the natural tendency of muscle to contract when separated from its attachments.

To facilitate draping the muscle down into the cavity, the bone is removed to a maximum extent in the area of the zygoma, and the bony ridge between the tegmen and the squama is rounded off. The posterior root of the zygomatous arch may also be removed. Cutting the temporal fascia across the base of the pedicle flap allows the muscle to stretch without interfering with the blood supply. Some of the edema in the flap can be removed by packing it into the cavity for a few minutes with adrenalized cotton packs.

Melted paraffin,* just before it begins to solidify, is now dropped with the aid of a medicine dropper into the middle ear until it forms a solidified plug of paraffin which rises to the level of the horizontal semicircular canal and covers the mouth of the Eustachian tube opening. When the paraffin is cooled it is then tamponaded to form a smooth filling of the middle ear cavity, avoiding any dead spaces.

A free split thickness graft from the patient's thigh or abdomen is then obtained for grafting the cavity.

The temporal muscle pedicle flap is brought down into the cavity to bridge the paraffin filling the middle ear space and to cover the horizontal semicircular canal and floor of the mastoid. The anterior pedicle skin flap is placed over the upper part of the muscle. The remainder of the cavity is skin grafted using separate pieces of skin, teased into place, over the tegmen, the sinus, the inferior spine and floor of the

*Tissue Mat Wafers, Fisher Scientific Co., New York, N. Y.

external canal. One long strip of skin is placed over the temporal muscle flap to cover its entire length. More paraffin is then poured into the mastoid cavity filling the entire bowl to the level of the mastoid cortex. The superior endaural incision is sutured and the endaural opening packed with ¼ inch strip gauze, turning the previously prepared posterior skin flap over the posterior edge and under the auricle. To prevent a hematoma from forming, a pressure pack is placed just above the auricle to cover the dead space created by raising the muscle flap.

Postoperative Interval Between First and Second Stage.

In the normal course of events there is drainage around the paraffin prosthesis in the mastoid cavity for about two or three weeks. Following this the drainage ceases. The paraffin is not disturbed until about six weeks postoperatively. With a dental elevator it is then worked out of the cavity through the endaural opening, in two or three large pieces. At this time the cavity should be well epithelized and dry. After observing the cavity for another two to four weeks the patient is booked for the second stage.

II. Musculoplasty, Second Stage.

A superior endaural incision is made extending upward, close to the helix, to a level just above the auricle. This allows the auricle to be laid back far enough to permit the division of the skin lining the bowl of the mastoid from that lining the undersurface of the auricle. This incision is made at the level of the cortex. The auricle is then retracted until there is a wide exposure of the mastoid bowl with its lining epithelium. Elevation of the skin begins at the posterior edge of the mastoid bowl toward the middle ear. As the skin is elevated, V-shaped wedges are removed superiorly and inferiorly to form a long, wide flap extending from the false drum over the middle ear. This flap should be very wide, and long enough to cover a large middle ear space and to allow for contraction of the tissues. The flap is elevated to expose the horizontal semicircular canal and the paraffin of the middle ear. Since mucosa has grown from the mouth of the Eustachian tube to envelope the paraffin, as skin is raised to the

middle ear the mucosal sheath must be incised to expose the paraffin. With a dental elevator the temporary paraffin prosthesis is rolled out of the middle ear cavity, revealing a middle ear space completely lined with newly grown mucosa. The flap is retracted anteriorly and a fenestra made over the surgical dome of the horizontal semicircular canal. The lavage-free cupola technique of Lempert is followed. The previously prepared flap is then replaced to seal the middle ear, to cover the fenestra, and to cover the floor of the mastoid cavity. The cavity is packed with paraffin mesh gauze and the superior endaural incision closed with several interrupted skin sutures.

SUMMARY.

The use of a temporary paraffin prosthesis to regrow mucosa in a restored middle ear cavity is described as part of the surgical technique of musculoplasty. The development of a controlled method for re-establishing a mucosal lining, in a re-created middle ear air-space, provides the last facet of technique needed for a predictable, standardized operation to restore hearing in chronic otitis.

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SOME ASPECTS OF THERAPEUTICS IN SINUSITIS.*†‡

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The author has been interested in analysis of the value of chemotherapeutic agents in the treatment of acute sinusitis and has collaborated in this work with Dr. Richard Reynolds and Dr. Leighton Cluff of the Department of Medicine of Johns Hopkins University School of Medicine.

The current therapy for acute sinusitis is directed towards the establishment of adequate drainage from the paranasal sinuses by the use of local decongestants, antihistamines, and the irrigation of the larger sinuses, when necessary.

Chemotherapeutic agents are felt to be of no value in the control of viral infective agents; however, in the control of bacterial invaders, the antibiotics are accepted as effective, and enjoy general use. The actual effect of the antibiotics in acute sinusitis remains undetermined, particularly since so many drug-resistant organisms have entered the bacteriological picture.

Let us review the use of antibiotics in the treatment of acute sinusitis for the past 20 years: In the 1944 Journal of Pediatrics, George Livingston[1] noted that the clinical course of sinusitis lasted only three to four days after administration of the sulfonamides, whereas the average duration of the ill-

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ness prior to the antibiotic era was three to four weeks. A review of other early reports of sulfonamide and penicillin therapy for sinusitis finds the main topics of interest to be the toxic effects of the sulfonamides and the debate as to whether the local route of medication was better than the systemic. A review of the medical literature from 1939 to 1960 fails to demonstrate any adequately controlled studies which correlate the type and incidence of infecting organisms, their antibiotic sensitivities, and the response of the bacterial sinusitis to chemotherapy.

In September, 1958, a study was begun by the Departments of Medicine and Otolaryngology of the current bacteriology, drug sensitivity, and therapeutic results with antibiotics in sinusitis. Acute maxillary sinusitis was chosen because of the ease with which clinical observation and treatment could be performed. Those individuals who had had prior sinus surgery were excluded from the study on the basis that their sinus membranes might be abnormal; no other contraindications were made. When the diagnosis of acute maxillary infection was established by the presence of antral clouding to transillumination and by the observance of purulent discharge from the affected sinus ostia, initial cultures were obtained from the drainage or from the antral washings, if irrigation was performed. Organisms recovered from the cultures were tested for antibiotic sensitivity. All coagulase-positive staphylococci were phage typed.

Patients were treated in serial order with one of three drug programs: *1.* erythromycin proprionate—500 mgm. every six hours, *2.* tetracycline—500 mgm. every six hours, and *3.* Vi-cillin-K 400,000 units every eight hours. These dosage schedules are about twice the usual therapeutic doses and were used on the advice of Dr. E. K. Marshall of the Department of Pharmacology. The drugs were administered for a period of seven days, and longer if necessary. Two cases on tetracycline developed severe gastrointestinal symptoms which necessitated discontinuance of the drug. Three cases on erythromycin proprionate required halving of the daily dosage because of epigastric distress. Any supportive therapy, except

the use of steroids, was permitted. Antihistaminic drugs and irrigation of the infected antrum were used at the discretion of the attending rhinologist; thus, most of the patients received nasal decongestants, anodynes, and one antibiotic.

Table I shows the initial signs and symptoms of the sinusitis study group. All of the cases presented with purulent nasal drainage, ethmoiditis, and clouding of the infected antrum to transillumination. A majority noted facial pain and facial

TABLE I.

Presenting Symptoms in 22 Cases of Acute Maxillary Sinusitis.

Signs and Symptoms	Number of Cases
Purulent Nasal Drainage	22
Facial Pain	14
Facial Tenderness	12
Ethmoid Sinus Infection	22
Frontal Sinus Infection	1
Facial Swelling	2
Fever	5
Chills	1
Deflected Nasal Septum	9
Previous Sinusitis	14
History of Allergy	5
Clouding of Antrum to Transillumination	22

tenderness. Sixty-four per cent gave a history of a previous sinusitis. Forty-one per cent were found to have a significantly deflected nasal septum.

Table II presents the relative incidence of bacterial organisms recovered from the initial culture of the nose, nasopharynx, or antral washings. The predominant organism was the coagulase-positive Staphylococcus aureus, which was present in 15 cases (68 per cent). Two of these were type 80/81 from hospital personnel; one was type 7/VA4, and the remainder were non-typeable coagulase-positive organisms. The pneumococcus was the second most common bacterium, which was present in eight cultures. Four of the sinusitis cases had mixed cultures of pneumococci and coagulase-positive staphylococci. The remainder of the initial culture studies is shown in the table.

Table III summarizes the relationship between the anti-

biotic used, the sensitivity of the organisms obtained on initial culture, and the clinical course of the infection. This bar graph demonstrates the actual number of cases in each category. The organisms are grouped together in this chart, as their specific drug sensitivities are presented later in the discussion. The *in vitro* sensitivities were established with a five-microgram disc for erythromycin and tetracycline, and a two-unit disc for penicillin. The results are also shown in this table. A clinically improved case is defined as one with maxillary sinusitis and ethmoiditis which showed marked

TABLE II.

Incidence of Organisms Obtained from Initial Culture.

Organisms	Number of Cases	
Hemolytic Staphylococcus Aureus	7	(Two Type 81 from Hospital Pers.) (Remainder Not Typeable)
Hem. Staph. Aureus with Pneumococci	4	(One Staph. Type 7/VA 4)
Hem. Staph. Aureus with Klebsiella	2	
Hem. Staph. Aureus with Type A B-Hem. Streptococcus	1	
Non Hem. Staph. Aureus	1	
Pneumococcus	2	
Pneumo. with Hem. Staph. Albus	1	
Pneumo. with a-Streptococcus	1	
Strept. Fecalis with Klebsiella	1	
Proteus	1	
E. Coli	1	

improvement of symptoms within seven days, and disappearance of signs and symptoms of infection within two and one-half weeks. All cases unimproved by seven days were classed as failures.

Every case but one treated with erythromycin proprionate was improved in seven days and clinically well within two and one-half weeks. All of the organisms in this group were erythromycin-sensitive except the one failure case which was a resistant Proteus. The tetracycline group cultured 54 per cent drug-sensitive organisms. Eighty-one per cent was improved in seven days and clinically well in two and one-half weeks, except for two failures which occurred in one individual with a drug-sensitive pneumococcal infection and in

another with a mixed drug-sensitive pneumococcal plus a drug-resistant staphylococcal sinusitis. Sixty-seven per cent of the penicillin group was drug-sensitive organisms. The penicillin treated cases cleared equally well except for one failure in a drug-sensitive pneumococcal sinusitis. At first glance, erythromycin proprionate appeared to have given better results than the other drugs; however, there was no apparent correlation between drug sensitivity and the ulti-

TABLE III

RELATION OF THE ANTIBIOTIC TO IN VITRO SENSITIVITY TESTS AND CLINICAL RESULTS

ANTIBIOTIC	NUMBER OF CASES	
ERYTHROMYCIN PROPRIONATE	SENSITIVE	R.
	CLINICALLY IMPROVED IN 7 DAYS	F.
TETRACYCLINE	SENSITIVE	RESISTANT
	CLINICALLY IMPROVED IN 7 DAYS	FAILURE
VI-CILLIN-K	SENSITIVE	RESIST.
	CLIN. IMP./ 7 DAYS	F.

R. = RESISTANT
F. = FAILURE

mate outcome of the infection. All cases of acute maxillary sinusitis cleared within two and one-half weeks, including the four original failures who were shifted to a different antibiotic. Two possibilities were apparent from the data shown in Table III. First, the *in vitro* drug sensitivity tests may not have been a reliable indication of the effectiveness of the drugs. Second, the drugs used may or may not have exerted a beneficial effect upon the course of the disease.

Subsequently, two studies were initiated: the first was a study of the normal bacteriological flora of the nose and

nasopharynx. Sixty-six individuals from the out-patient clinics of the Johns Hopkins Hospital with essentially normal nose and throat examinations received nasal and nasopharyngeal cultures. Only two of these cultures were negative for aerobic bacteria; 48 individuals grew single organisms, and 16 grew multiple organisms. The incidence of coagulase-positive staphylococci and pneumococci is shown in Table IV.

Twenty-seven per cent of the normals had cultures containing coagulase-positive staphylococci as compared with 68

TABLE IV.

Incidence of Coagulase-Positive Staphylococci and Pneumococci in the Study Groups.

Organism	Normal Group	Sinusitis Group
Coag.-Pos. Staphylococci	27% (18/66)	68% (15/22)
Pneumococci	18% (12/66)	26% (8/22)

TABLE V.

Antibiotic Sensitivity of Coagulase-Positive Staphylococci in the Study Groups.

Sensitivity	Normal Group	Sinusitis Group
Penicillin Resistant 2 unit disc	44% (8/18)	67% (10/15)
Tetracycline Resistant 5 mcgm. disc	11% (2/18)	50% (7/15)
Erythromycin Resistant 5 mcgm. disc	0% (0/18)	7% (1/15)

per cent in the acute sinusitis study. The incidence of pneumococcal organisms in the normal group was 18 per cent as compared with 36 per cent in those individuals with sinusitis. The 18 per cent figure for normals compares favorably with an average figure of ten per cent in other studies.[2] The table shows a much higher incidence of staphylococci and pneumococci in the sinusitis group, although the total incidence of staphylococci of all types is about the same for both groups, since the incidence of coagulase-negative staphylococci in the normal group was 35 per cent (23/66).

Analysis of the drug sensitivities reveals some interesting comparisons, as shown in Table V, which compares only the

coagulase-positive staphylococci cultures. Sixty-seven per cent of the sinusitis group was penicillin-resistant as compared with 44 per cent in the normals. Fifty per cent of the sinusitis cases was tetracycline-resistant as contrasted with 11 per cent of the normals. Seven per cent of the sinusitis cases was erythromycin-resistant as compared with 0 per cent of the normals. More drug-resistant organisms were present in the sinusitis cases, but in both groups, erythromycin showed the best *in vitro* inhibition of bacterial growth, with tetracycline and penicillin showing decreased activity in that order.

As might be expected, those individuals with acute maxillary sinusitis had a larger incidence of pathogens isolated in their cultures, and these pathogens demonstrated a higher percentage of drug-resistant forms. The increased incidence of drug-resistant strains of bacteria in the patients with sinusitis may be attributed to previous treatment with antibiotics, as many individuals of this group gave a positive history of previous attacks of sinusitis. It is interesting to note that 27 per cent of the normal group also had coagulase-positive staphylococci in their cultures. This finding suggests the possibility that individuals sustain a bacterial infection with organisms already present in their nose or nasopharynx, when damage to the surface epithelium occurs as the result of viral infection, allergic reaction, and so forth.

The other part of the second study is the establishment of a double-blind evaluation of erythromycin proprionate and a placebo in the treatment of acute maxillary sinusitis. The criteria are the same as those used in the initial comparative drug study. Supportive therapy, including decongestants, antihistamines, and antral lavage is used in both the treated and control groups. The initial number of cases is too small to make any conclusions at this time, except to note that sinusitis in many of the placebo-treated cases has cleared rapidly with supportive therapy alone—which once more emphasizes the importance of promoting adequate sinus drainage.

We do not advise the otolaryngologist to discontinue antibiotic therapy for sinusitis, although some of the observations just given seem to question the value of chemotherapy. In

the pre-antibiotic era, the majority of acute sinusitis patients recovered spontaneously on supportive therapy. Early reports of antibiotic treatment of upper respiratory infection gave glowing statements that the infections disappeared within two or three days. The present study showed disappearance of acute maxillary infection in a period of seven to 20 days, regardless of the antibiotic administered. One-half of our failures occurred with drug-sensitive organisms. These observations lead to the conclusion that drug therapy may be less important in the therapy of acute sinusitis today than it was fifteen years ago.

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INSTITUTE ON OCCUPATIONAL HEARING LOSS.

This course presented by Colby College, Waterville, Maine, for nine consecutive years is designed to train physicians interested in this problem, nurses, plant engineers and others, in initiating and conducting hearing conservation programs in noisy industries. The curriculum includes discussion of the causes of occupational deafness, but is primarily concerned with its prevention. In order to allow for maximum individual instruction, course enrollment is limited. The diversity of experience of the faculty members, assembled especially for this course, provides an unusual opportunity for personal consultation on any aspect of occupational hearing loss.

The dates of the course are August 14-19 and the fee of $200 includes tuition, board and room. For further information write to William A. Macomber, Colby College, Waterville, Maine.

A SIMPLIFIED AUDITORY TEST FOR INFANTS AND YOUNG CHILDREN.*

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I.

The past decade and a half has witnessed the generation of widespread interest and spectacular progress in the development and refinement of techniques for differential diagnosis of hearing disturbances among children with delayed speech behavior.[1,3,4,5,9,10,16,21,24,26,28,29] Organic hearing loss and late speech development present an interdependent sequence in child growth, but the realization that failure to develop speech may be contingent on many other etiological factors besides hearing loss, has universal acceptance. When hearing acuity can be demonstrated the causal factor may be explored through case history and other appropriate studies. In recent years careful study has revealed that many children with normal threshold acuity behave as non-hearing children. Increasing research supports the hypothesis that young children with communicative disorders do not permit facile diagnosis.[3,4,5,15,16] A major and crucial problem has been to determine whether these children present true symbolic disabilities, whether the communicative processes have been contaminated by failure to develop simple discriminatory behavior, or whether the input auditory processes are so disturbed physiologically that auditory recognition or identification, memory, recall, and synthesis cannot be consummated. Most of these children may be identified by at least four characteristics: *1.* lack of auditory sensitivity, *2.* in-

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ability to speak or communicate, *3.* difficulty with learning processes, *4.* behavior disturbances. As children grow older, psychological concomitance of frustration, anxieties, and inadequate learning, further complicate the identification of these children. A simplified but reliable test which would specify the stimulus and identify the response for eliminating organic hearing loss before the psychological manifestations influence children's responses, should expedite medical and educational rehabilitation.

II.

Experience during World War II with the sudden onset of nonorganic hearing loss among soldiers under distress and anxiety, alerted the medical and educational professions that similar phenomena might be operating among different populations. The development of audiometrics, speech tests, EDR, and EEG techniques proved satisfactory with the adult population. The belief that these procedures could be extended unequivocally in identifying children has proved disappointing. EDR audiometry appears to be unsuited for most hyperactive children and extremely traumatic for some. It is difficult enough to test young children who cooperate or participate actively in the test procedures especially below the age of three. The testing of children under two would be more desirable since the psychological manifestations that interfere with accurate testing do not appear full blown before this age; still, testing such children with regular audiometric techniques proved to be unfeasible.

Previous investigators have searched for a simplified test for infants and young children. Froeschels,[12,13] studied motor responses of newborns to sound stimuli. Children between the age of one-half and nine days of age were employed in the study. Tuning forks and whistles were utilized as the stimuli. The investigation revealed only one child responded to tuning forks, while the remaining children responded to percussion instruments. The most commonly observed response appeared to be the acousticopalpebral reflex. Some children moved their eyes and others their heads towards the

stimuli. The Ewings,[8,9,10] devoted many years to the investigation of hearing responses of children. They observed children's responses to different sound sources and postulated the presence of the auropalpebral reflex and localization as indication of threshold hearing among young children. They also observed that children with hearing losses rarely localized. They extensively explored testing procedures among children and even developed a developmental schema for auditory responses. Their studies as well as Froeschels' suggest the inadequacy of the tuning fork and regular audiometry as tests for functional hearing for infants and young children. The Ewings' research indicates the most common responses of children from three to six months of age are reflexes and localization as indicating threshold acuity. With growth the reflexes disappear while responses due to learning activities become more prominent. They believe in the efficacy of the voice as a test source; furthermore, the Ewings exhibited a good deal of concern if the child did not manifest responses to auditory stimuli by the end of the first year. Their tests were informal and subjective but contained neither an exact specification of the stimulus nor a quantification of the response. Griffiths,[17,18] comprehensively investigated the abilities of babies and constructed a developmental continuum of the different modalities. Her work tends to support the concept that hearing functions as a prime modality reflecting growth and that hearing development is inextricably interwoven in the matrix of the over-all growth.

Dix and Hallpike[6] formulated a test for obtaining auditory thresholds for children. Their test employed a peep-show technique. With children who do not present problems the method works very satisfactorily; however, for young children the application of the test proves restrictive, since there is a question as to whether children below two years of age can comprehend and execute the necessary responses. Keaster[21] devised a method for testing young children employing words. This method possessed the distinction of measuring the hearing responses in 2 db steps. The children must, nevertheless, know the words, and for hyperactive, distractible,

and supposedly "aphasic" children the method provides little information concerning hearing acuity.

Wedenberg[29] experimented with an auditory test for detecting deafness among newborn infants. He capitalized on the appearance of the APR reflex and attempted to determine the relationship between the reflex and threshold obtained by the APR, by observing another acoustic reflex that grossly resembled the APR reflex, specifically the stapedius reflex. He suggested a very high correlation between these two reflexes in their ability to wake children from sleep with a stimulus of 70 plus or minus 5 decibels. Children awakened by this stimulus level he considered as possessing normal threshold hearing. This value of 70 plus or minus 5 decibels he considered as the maximum intensity possible since at higher intensity levels non-hearing infants with recruitment might theoretically be awakened. The test appears to be grounded on a theoretical basis and may possess face validity; nevertheless, it needs empirical substantiation.

In 1948, Bordley, Hardy, and Richter,[1] described an audiometric technique involving the use of the galvanic skin resistance response. They systematized the procedure which in the hands of the Johns Hopkins team proved very efficient and gained wide attention, so that many clinics in this country now include EDR equipment as part of their instrumentation for testing. Doerfler[7] studied the use of neurophysiological clues in determining auditory acuity and concluded that EDR procedures offered a very promising approach in the detection of children difficult to test with other methods.

Examination of the literature discloses work embracing GSR responses as early as 1937. Hovland[19] studied conditioned responses with varied frequencies of tone in experiments using conditioning galvanic skin reaction. He was interested in relating his findings to experiments in learning, especially in the areas dealing with generalization and extinction. Ellson[11] studied the spontaneous recovery of galvanic skin response as a function of recovery interval. He successfully isolated the following variables: *1.* the expectation of shock

did not show a progressive alteration, *2.* negative adaptation was influenced by the amplitude of response.

Later, Humphreys[20] investigated the extinction of psychogalvanic responses following two conditions of reinforcement and concluded from his data that the strength of the conditioned responses correlated with the strengths of the reflexes. He also observed a striking similarity between magnitude and adaptation. Littman[23] also studied the conditioned generalization of galvanic skin reaction to tones. His findings supported Hovland's[19] conditioning generalizations. In addition, he pointed out at least two problems of generalization requiring further study. The problems of causal mechanisms and the effect of generalization on other behavior did not appear to be clearly delineated. Wickens[30] later experimented with primary stimulus generalization of GSR under two conditions using tones as the stimulus. His results show that extinction curves became progressively steeper with increased extinction rather than concave. He also reported that primary stimulus generalization is actually bell-shaped and suggested that the function varied with the number of parameters. His study also indicated that absolute auditory threshold intensity was accurately measured at the autonomic level. Noble[27] explored conditioned generalization of the galvanic skin response to a sub-vocal stimulus. He hypothesized that GSR conditioned to a temporal stimulus compound, *e.g.*, light and own voice, could be generalized to a sub-vocal stimulus. If this proved to be true it would show sub-vocal voluntary control over a previously involuntary response. Girden[14] investigated the role of "set" in human conditioning under conditions of awareness by the subject. His results show absolute auditory threshold intensity accurately measured at the autonomic level with or without the subject's conscious cooperation. Moeller[25] tried to determine whether or not optimal CS-UCS interval in GSR conditioning is the same for the autonomic and the central nervous system. White and Schlosberg[31] studied the degree of conditioning of GSR as a function of the period of delay. He found some significance in the two-second interval response.

Goodhill, Rehman, and Brockman[16] investigated objective skin resistance audiometry. They purported to find a completely objective audiometric technique. Their results illustrated a moderately stable base line could be procured in testing normal children and adults. Their records for infants under 20 months of age were incomplete and variable. Their study permits the speculation whether or not GSR can be used with children with central nervous system disabilities and raises the question as to whether the nervous system of infants is sufficiently stabilized to permit interpretation. An evaluation of GSR research reflects many theoretical difficulties. The questions raised in the discussion suggest that these variables and factors are still inherent in the EDR approach to testing the hearing of young children.

The eventual disappointment with EDR audiometry as an infallible technique for detecting hearing loss compelled other investigators to search for a more efficient method for the testing of difficult children. Marcus, Gibbs, and Gibbs[24] used EEG audiometry as a potential instrument and method for testing the hearing of very young children. They felt that they could illustrate the nonspecific arousal response elicited by the stimulation of tactile, olfactory, and visual modalities as applying to the auditory modality as well. The children in their study had undergone all of the functional and organic tests for hearing. The children were placed under induced sleep. The method was abandoned because the audiometer circuit interfered with the electroencephalographic records. They had difficulty with shielding and grounding and these introduced many artefacts into the tracings. Instead of the audiometer they employed tuning forks which proved to have insufficient intensity to be effective. They introduced high intensity level sounds, and the arousal responses were recorded on the EEG tracings. The application of these sound stimuli could not provide information on threshold acuity; nevertheless, the idea had merit and was later tested again. As early as 1939, Davis, *et al.*,[2] studied the electrical reaction of the human brain to auditory stimulation during sleep. This was one of the first attempts to obtain auditory acuity by EEG. Marcus, Gibbs, and Gibbs[24] had attempted to use EEG

audiometry to diagnose early the hearing of infants suspected of organic hearing impairment. Derbyshire and McDermott[3] refined and extended the EEG method for evaluating auditory dysfunction in children; furthermore, they formulated a method of interpreting and evaluating the audiogram as reflected in the EEG tracings. They found four component parts of the total response could be identified: *1.* "k" formation, *2.* on-effects, *3.* off-effects, *4.* late effects. They arbitrarily set their criterion of 50 per cent of identifiable responses at minimum threshold level, as the auditory threshold. In addition, they devised a procedure whereby these responses might be objectively identified. They postulated that EEG response in sleep and alpha suppression components suggested an alerting mechanism of the reticular formation. This mechanism required maximum alertness in attending to auditory cues. This procedure embraces some very great advantages over other methods. Distractible, hyperactive, and anxious children can be controlled by sedation. If the response proves satisfactory the auditory status may be clearly identified. Once the threshold acuity has been established other aspects of the child's development are pursued. Derbyshire is still exploring and refining the EEG technique. This approach offers great promise for future application; nevertheless, the method still possesses some disadvantages: *1.* children have to be sedated, *2.* procedures are lengthy, *3.* interpretation of the audiogram requires specialists. These disadvantages are minor and may be effectively overcome by the establishment of teams. Insofar as it gives information which other methods fail to provide, the disadvantages merely become inconveniences.

III.

This experiment was designed for the purpose of discovering a simplified auditory test for infants and young children. The criteria for the test should be: *1.* short in duration. *2.* easy to administer, *3.* sufficiently reliable as to be practical as a screening technique. The instruments employed were adapted to provide two signal sources, recorded disc material

and a white noise source. The phonograph pickup for recorded presentation was a G.E. reluctance pickup with a preamplifier equalized to produce a response meeting the standards of the N.A.R.T.B. The white noise was produced by a conventional gas discharge tube fed through an equalized amplifier. The resultant noise spectrum was flat within three plus decibels from 40 to 12,000 cycles. Each signal source passed first through an equalized preamplifier, then a 20 decibel pad which could be switched in or out. Next, the signal passed through a 50 Watt power amplifier and into the distribution and metering system. At this point the meter could be switched to either signal source to establish the calibration. Each signal source was then fed to a 20 decibel pad which could be switched in or out and then to four variable pads for the speech and four for the noise, each representing the respective speakers in the four corners of the room. Each of these pads was a wire-wound unit having about 35 decibels attenuation, and each had a key to feed either its respective speaker or a dummy load.

Four pairs of speakers with an azimuth difference of 90° were situated inside the test chamber. Each speaker was a high fidelity six-inch permanent magnet type mounted in a cabinet of the Helmholtz resonator type. Each cabinet was 14 inches high, 11 inches wide, and 10 inches deep. Each pair of speakers was supported by a wood base with the cone of the noise speaker being eight inches vertically superior to that of the recorded speaker and approximately at ear level of the seated subject. The four speakers of each set were inphase, being electrically phased through the application of direct current and observation of the direction of cone excursion. The system of attenuators and keys made it possible for the eight speakers to be used singly or multiply through any of the possible combinations of speakers. The attenuation in all cases was continuous, readable in one decibel steps, with a range of 20-100 decibels (sound pressure level in microbars). The SPL's obtained for any of the testing in the test chamber were those measured in the center of the testing area (87 inches from each of the pairs of speakers). A small pendulum was suspended from the ceiling at this central point

and in testing, the subject was seated on the mother's lap so that the pendulum was directly over the center of the head.

Figure 1 presents a schematic arrangement of the equipment and the test chamber. The test chamber was constructed to provide a sound field. The acoustic properties of this test chamber are such that the ambient noise level within the

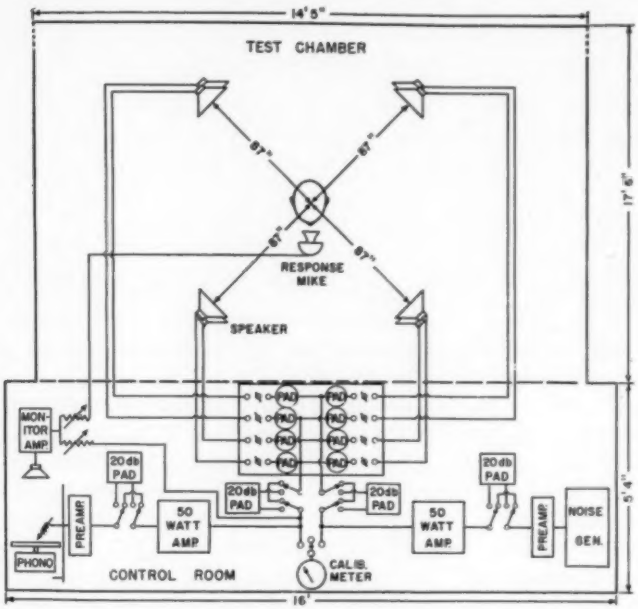

Fig. 1. Sound field and control room with schematic and instrumentation used for Speech Reception Thresholds and Discrimination Scores.

room is less than 24 decibels, as measured by the General Radio Sound Level Meter set at position A. For the purposes of this experiment, the reverberation effects of the test chamber were further improved by hanging a three-inch blanket of fiber-glass upon the walls.

Figures 2 and 3 show the acoustic properties of the test

chamber (when unoccupied). These characteristics were determined in two ways:

1. Evaluation of the ambient noise level with regard to the spectrum;

2. Evaluation of the absorption qualities of the sound treatment by comparing the actual Sound Pressure Level (re .0002 dyne/cm.^{2}) to that expected by use of the inverse square law.

The ambient noise level was measured with a General Radio 1551A Sound Level Meter and 760B Sound Analyzer, a Hew-

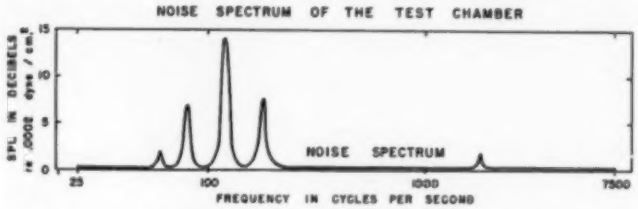

Fig. 2. Noise spectrum of test chamber.

lett Packard Model 130A Oscilloscope, and a Ballantine voltmeter. The use of the equipment provided a means of distinguishing between the noise indications within the test equipment and the ambient noises within the room. For this evaluation, the Sound Level Meter was placed at the center of the test area, while the Sound Analyzer and Oscilloscope in the control room were connected to the Sound Level Meter in the testing room. With the Sound Level Meter set at position A, the ambient noise spectrum in the room (after adjustment for equipment noises had been calculated) consisted of the components indicated.

In the comparison of the actual Sound Pressure Level (SPL) to that predicted under perfect conditions by the inverse square law (decay characteristics of the room), measurements were made at distances of 1, 2, 4, 8, and 12 feet from each of two speakers. A Western Electric Model 640-AA condenser microphone (and cathode follower), Grayson Stad-

ler Condenser Microphone Complement Model 726-A, and Ballantine Logarithmic scale voltmeter were used in measuring a constant output of white noise at the distances indicated above. The condenser microphone and cathode follower were mounted upon a microphone stand at a height of approximately ear level. The Condenser Microphone Complement and the voltmeter were placed near the test chamber window. The readings were observed from the control room. At none

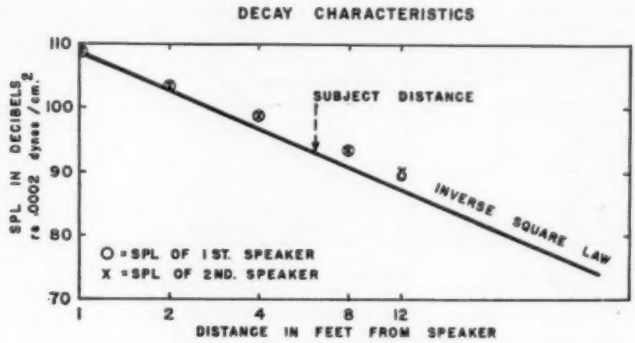

Fig. 3. Decay characteristics of the test chamber.

of these distances was the measured SPL more than two decibels higher than the theoretical (inverse square law) SPL.

Figure 4 shows an 11-month-old child in position for testing. The tester could observe the child in the testing chamber through a one-way vision mirror. He could observe his responses and also read the input sound pressure levels directly from the meter. The materials utilized were: music with slow rhythmic tempo of the lullaby type, and also the tempo of march. White noise and recorded pure tones were also used. The frequencies employed ranged from 250 to 6000 cycles. In addition, voice materials were recorded for presentation. The presentation of stimuli was very short in duration. If the child would not remain alone in the test chamber he was permitted to sit in his mother's lap to eliminate and control the fear and anxiety components. The stimuli were presented

in random order. The child faced the one-way vision mirror so that the examiner could observe his reactions. There were no distractions presented.

Subject.

Fifty children, ranging from ten months to three years of age, were employed in the experiment. Table I indicates the different ages. The total test took less than five minutes.

Fig. 4. Eleven-month-old child in position for testing.

IV.

Of the 50 children, 31 were found to have normal hearing when compared with audiograms obtained several years later. The remaining 19 children were found to have hearing losses.

Table II presents the distribution into categories of the 50 children. Category I represents hearing threshold from 0 to 20 decibels, II—20 to 40 decibels, III—40 to 60 decibels,

TABLE I.

Subject	Age First Testing	Speech First Testing	Hearing Loss Suspected	Localization Response	Referral Source	Age Retested		Audiogram Cycles 500	1000	2000	Speech Status	Final Diagnosis
CL	2 yrs.	None	Yes	20 db definite	Medical	4	R	0	0	0	Some words	E.D.
							L	0	0	0		
PR	15 mos.	None	Yes	60 db startle	Medical	4	R	0	0	0	Echolalia	M.R.
				20 db definite			L	0	0	0		
TM	18 mos.	None	Yes	60 db startle	Medical	4	R	0	0	0	Echolalia	M.R.
				Cry			L	0	0	0		
				20 db definite								
DB	18 mos.	None	Yes	70 db startle	Medical	4	R	0	0	0	Phrases	D.M.
				20 db definite			L	0	0	0	Sentences	
RLB	2 yrs.	Words	Yes	60 db startle	Medical	5	R	0	0	0	Speech within	D.M.
				10 db definite			L	0	0	0	normal limits	
RT	20 mos.	None	Yes	100 db awareness	Medical	5	R	70	90	95	Sounds	H.L.
							L	70	90	90		School for Deaf
DK	26 mos.	None	Yes	90 db no startle	Medical	5	R		10	EDR	Grunts	M.R.
				20 db definite			L		10	EDR		
HC	2 yrs.	None	Yes	100 db awareness	Medical	4	R	70	80	95	Sounds	H.L.
							L	15	80	90		School for Deaf
DK	2 yrs.	None	Yes	15 db definite	Medical	5	R	0	0	5	Echolalia	M.R. + C.N.S.
							L	0	0	0		
DD	2 yrs.	None	Yes	100 db awareness	Medical	4	R	70	90	NR	Few words	H.L.
							L	65	90	NR		School for Deaf
LD	3 yrs.	Some words	Yes	80 db definite	Medical	5	R	50	60	70	Phrases	H.L.
							L	55	60	70	Sentences	School for Deaf
KC	26 mos.	Some words	Yes	70 db definite to left	Medical	5	R	75	95	NR	Words	H.L.
							L	50	60	65	Sentences	Hearing Aid
TF	3 yrs.	Some words	Yes	70 db definite to right	Medical	5	R	30	40	55	Sentences	H.L.
							L	60	65	75		Hearing Aid
PR	2 yrs.	Words	Yes	55 db definite	Medical	4	R	30	35	40	Speech within	H.L.
							L	30	40	45	normal limits	Hearing Aid
DA	2 yrs.	None	Yes	15 db definite	Medical	4	R	0	5	0	Sentences	E.D.
							L	0	0	0		
ES	2 yrs.	None	Yes	20 db definite	Medical	4	R	0	0	0	Echolalia	M.R. + C.N.S.
							L	0	0	0		
RC	2 yrs.	Some words	Yes	30 db definite	Medical	5	R	20	20	20	Speech within	H.L.
							L	15	20	20	normal limits	Hearing Aid

TABLE I Continued.

Subject	Age First Testing	Speech First Testing	Hearing Loss Suspected	Localization Response	Referral Source	Age Retested		Audiogram Cycles 500	1000	2000	Speech Status	Final Diagnosis
BD	2 yrs.	None	Yes	60 db startle	Medical	5	R	0	10	0	Echolalia	M.R. + C.N.S.
							L	0	5	0		
RL	3 yrs.	None	Yes	60 db startle	C.P. Center	6	R	0	0	5	Sentences	M.R. + C.N.S.
							L	0	0	0		
HA	3 yrs.	Some words	Yes	20 db definite	C.P. Center	5	R	0	10	5	Sentences	S.L.
							L	0	5	5		
DD	28 mos.	None	Yes	20 db definite	C.P. Center	5	R	0	0	0	Echolalia	M.R. + C.N.S.
							L	0	0	0		
JL	2 yrs.	None	Yes	100 db awareness	Medical	5	R	80	90	95	Words	H.L.
							L	80	90	95		School for Deaf
RG	11 mos.	None	Yes	70 db startle	Medical	5	R	0	0	0	Echolalia	M.R. + C.N.S.
							L	0	0	0		
NC	2 yrs.	None	Yes	70 db startle 20 db definite	Medical	5	R	0	0	0	Echolalia	M.R. + C.N.S.
							L	0	0	0		
MR	10 mos.	None	Yes	100 db awareness	Medical	4	R	70	85	95	Some words	H.L.
							L	75	85	95		School for Deaf
JW	3 yrs.	Words	Yes	20 db definite	Medical	5	R	0	0	0	Phrases and	S.L.
							L	0	0	0	sentences	
DS	28 mos.	Words	Yes	15 db definite	C.P. Center	5	R	0	0	5	Sentences	E.D.
							L	0	0	0		
LAL	3 yrs.	Words	Yes	60 db definite	Medical	5	R	30	35	40	Sentences	H.L.
							L	35	35	45		Hearing Aid
MS	3 yrs.	Words	Yes	20 db definite	Medical	5	R	0	10	10	Sentences	M.R.
							L	5	10	10		
DM	2 yrs.	None	Yes	20 db definite	Medical	5	R	0	0	0	Words	M.R. + C.N.S.
							L	0	0	0		
SC	2 yrs.	None	Yes	20 db definite	Medical	5	R	0	0	0	Words	M.R.
							L	0	0	0	Phrases	
KMc	2 yrs.	None	Yes	60 db startle	C.P. Center	5	R	0	0	0	None	M.R. + C.N.S.
							L	0	0	0		
MDM	2 yrs.	None	Yes	60 db startle	Medical	5	R	0	0	0	Phrases	M.R. + C.N.S.
							L	0	0	0		
JZ	2 yrs.	None	Yes	100 db awareness	Medical	5	R	75	85	95	Words	H.L.
							L	80	80	95		School for Deaf

TABLE I Continued.

Subject	Age First Testing	Speech First Testing	Hearing Loss Suspected	Localization Response	Referral Source	Age Retested		Audiogram Cycles 500	1000	2000	Speech Status	Final Diagnosis
DLE	2 yrs.	Words	Yes	25 db definite	Medical	4	R	0	10	10	Speech within	D.M.
							L	0	5	0	normal limits	
ARF	3 yrs.	Grunts	Yes	15 db definite	Medical	4	R	0	0	0	Sentences	E.D.
							L	0	0	0		
SA	2 yrs.	None	Yes	80 db definite	Medical	4	R	40	55	70	Words	H.L.
							L	50	60	75	Phrases	Hearing Aid
LM	2 yrs.	None	Yes	70 db definite	Medical	5	R	45	55	60	Words	H.L.
							L	50	55	70		Hearing Aid
TM	2 yrs.	None	Yes	40 db definite	Medical	5	R	10	20	20	Sentences	H.L.
							L	10	15	15		
BF	2 yrs.	None	Yes	40 db definite	Medical	5	R	25	35	55	Sentences	H.L.
							L	25	40	55		Hearing Aid
VE	20 mos.	None	Yes	10 db definite	Medical	5	R	5	10	10	Words	M.R.
							L	0	10	10		
PK	22 mos.	None	Yes	40 db definite	Medical	5	R	20	30	35	Sentences	H.L.
							L	20	30	35		Hearing Aid
HLF	14 mos.	None	Yes	70 db startle	Medical	4	R	0	0	0	Grunts	M.R. + C.N.S.
							L	0	0	0		
GB	2 yrs.	None	Yes	15 db definite	Medical	4	R	0	0	0	Echolalia	M.R.
							L	0	0	0		
BH	16 mos.	None	Yes	70 db startle Cry 20 db definite	Medical	4	R	0	0	0	Sentences	E.D.
							L	0	0	0		
DL	11 mos.	None	Yes	20 db definite	Medical	4	R	0	5	0	Sentences	D.M.
							L	0	0	0		
CM	21 mos.	None	Yes	70 db startle 20 db definite	Medical	5	R	0	5	5	Echolalia	C.N.S.
							L	0	5	0		
DS	2 yrs.	None	Yes	80 db definite	Medical	5	R	55	70	80	Words	H.L.
							L	60	75	80		School for Deaf
SW	2 yrs.	Words	Yes	100 db definite	Medical	5	R	70	80	95	Words	H.L.
							L	70	85	95		School for Deaf
JR	12 mos.	None	Yes	20 db startle Cry 15 db definite	Medical	5	R	5	5	5	Words	M.R.
							L	0	5	5		

IV—60 to 80 decibels, V—80 decibels and above. Table III shows the disposition of the 31 children with a hearing threshold in Category I. Of the 31 children who were found to have normal threshold acuity, five proved to be emotionally disturbed, ten mentally retarded, one with central nervous system disorder, 11 with mental retardation plus central nervous system disorders, and four delayed maturation. Table I presents the final diagnosis of these children several years later.

Analysis of the results reveals the following responses were observed: Children with normal hearing exhibited the startle response at 90 decibels on first presentation and local-

TABLE II.

Distribution of the 50 Children Into Hearing Categories.

I	II	III	IV	V
31	2	6	3	8

TABLE III.

Distribution of the 50 Children Into Final Diagnostic Categories.

E.D.	M.R.	C.N.S.	M.R. + C.N.S.	D.M.
5	10	1	11	4

ized accurately up to 20 decibels. This finding appears to be inconsistent with other protocols. Myklebust[26] pointed out that children with central nervous system disorders were unable to localize. This finding was not substantiated in the study. On the contrary, the localization responses of the children in the study confirmed the Ewings' and the Froeschels' earlier findings concerning localization. Many children who responded with the startle at 90 decibels, also cried. Some of the children who gave the startle response also exhibited strong fear and anxiety reaction. The most reliable stimulus proved to be music; for most children with behavior and central nervous system disturbances, quiet rather than agitated music proved to exert a very quieting influence. Some of the children remained fixated, while others kept time to the music with the hands and feet. Music also elicited

emotional reactions from some children. The startle response was clearly indicated when the whole organism responded to the stimulus. Localization responses included movement of the eyes, head, and different parts of the body, toward the sound source. Among many of the children with central nervous system disturbances, adaptation occurred rapidly. The duration of the stimulus was controlled to avoid this. Landau, *et al.*,[22] suggest that the short duration of the stimulus proved less vulnerable for adaptation than a longer stimulus. Among other children the sudden onset of stimulus precipitated chain reaction release phenomenon. The spastic and athetoid children responded with a violent stretch reflex and involuntary movements involving the whole body. None of the children, including the severely hearing-impaired, proved to be insensitive to noise. The hearing-impaired children did not localize. They responded with attention and alertness without turning their heads. The sound seemed to bewilder them, and they gave evidence of an inability to cope with it. Some of the children exhibited fear behavior, attempting to escape the sound by pressing and escaping into the mothers' bodies. The expression of the eyes continued to reflect a scanning reaction.

White noise proved to be a very effective signal. Landau, *et al.*,[22] found this sound source effective in determining threshold acuity. He reported white noise and speech reception thresholds to be equivalent in obtaining threshold acuity in their study. The shortened presentation of the stimulus prevented adaptation and continued to alert the children. For children who responded but who had delayed speech, it was necessary to continue study for determining the causal factors of their nonverbal behavior. For children who might not respond in such a test, continued study is indicated. On the basis of the test results in this study, the presentation of stimuli to elicit localization and other concomitant responses from infants and very young children proved to be a simple, efficient, quick, and reliable test.

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THE CASE AGAINST ANTIBIOTIC PROPHYLAXIS IN MAJOR HEAD AND NECK SURGERY.*

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INTRODUCTION.

Antibiotic prophylaxis may be defined as administration of antibiotics to prevent rather than to treat infection. This report discusses antibiotic prophylaxis with particular reference to major head and neck surgery. The problem is of extreme importance not only to our own patients but also to the entire hospital population—all patients, doctors, nurses and employees—and eventually to the community as a whole because of the development of antibiotic-resistant bacteria in any or all of these reservoirs. While the past 15 years have seen the emergence and incredible growth of the science of antibiotics, we have also begun to find definite evidence that antibiotics are not going to abolish the problem of infection in surgery and indeed may often complicate it.[1]

TYPES OF SURGICAL WOUND.

In the process of obtaining statistics on the incidence of surgical infections, Meleney[3] classified surgical operations as *a.* Clean: aseptic wounds in which exposure to bacteria is only minimal and is inevitable, and adequate techniques are utilized. An example of this in neck surgery is the usual radical neck dissection. *b.* Contaminated: aseptic wounds in which there is unavoidable contact with potentially infective material in the operative field, other factors remaining unchanged. An example is total laryngectomy. *c.* Infected: wounds in which septic infection is present in the field at the time of operation, as in radical antrostomy for infected antro-oral fistula. Some 2000 cases were analyzed. The infec-

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tion rates with clean surgical wounds were between 2 and 3 per cent and with contaminated wounds were 8 to 12 per cent.

This survey was made before antibiotics were used clinically so that one may safely assume that the figures would not differ significantly today; thus, it might be said that the use of prophylactic antibiotic therapy for all patients seems useless in 97 to 98 per cent of clean cases and in 88 to 92 per cent of the contaminated cases.

BASIC FACTORS IN WOUND INFECTION.

The sole purpose of giving prophylactic antibiotic therapy is to prevent wound infection. Any particular wound infection, however, may be caused primarily by one or more basic factors, the major of which are:

1. Crude Surgery. The surgical "facts of life" are that rough surgery, bundle type of tissue ligation, heavy suture material, blunt dissection, heroic retraction and failure to treat soft tissues gently, will result in tissue necrosis. This will permit a relatively small number of organisms to grow and cause a wound infection. Failure to obtain complete hemostasis, to obliterate dead space, to drain adequately or to dress wounds properly will also increase the incidence of wound infection.

2. The Depleted Patient. The surgical patient with inadequate protein, fluids, blood, endocrine secretions or with impaired function of the liver, or gastrointestinal or urinary tract, or who is in shock from any cause loses his resistance to infection and may become a culture medium upon which bacteria may thrive.

3. Massive Bacterial Contamination. This results from failure of asepsis, from improper technique regarding preparation of the patient's skin, failure to sterilize instruments and operating room equipment or from flagrant violation of rules forbidding those with respiratory infections, furuncles and so forth from coming into contact with patients in any area of the hospital, especially the operating room. Also included in this group is the infectivity potential of the hospital

with regard to the number of bacteria present on beds, curtains and laundry and in the air generally. Kitchen and morgue also must not be forgotten in this regard.

4. Virulent Bacterial Contamination. When bacteria are toxic enough to necrose tissue and produce their own favorable substrata, the property of virulence exists. The virulent organism is usually a staphylococcus, a Beta streptococcus, or rarely a clostridial organism. Prophylactic antibiotic therapy which would sterilize an operative field and prevent the growth of bacteria presents the only logical basis for the use of drugs in this way; however, we still may not be able to sterilize the surgical field by giving antibiotics at this stage. The Beta streptococci remain very susceptible, but the other organisms may not be responsive or may require a combination of antibiotics, and even then, prophylaxis might not succeed.

It must be emphasized in this regard that the reservoir of the virulent organism is frequently a member of the operating team. Nahmias *et al.*[4] recently reported a series of 45 thoracic surgical cases; all of the patients were given prophylactic antibiotics, and in 15, significant wound infections developed which by phage typing were traceable to one surgeon.

THE THERAPEUTICS OF PROPHYLAXIS.

Antibiotics, like surgical wounds, have varying characteristics. Antibiotics, such as penicillin, are bactericidal, but most wide-spectrum antibiotics are bacteriostatic. Penicillin frequently causes severe reactions and, therefore, the tendency has been to rely less on penicillin and more on the wide-spectrum drugs for prophylaxis. Here the problem of which drug or combination of drugs to use must be faced. With prophylaxis, a culture is not available on which to base our therapy and, as has been pointed out many times, the use of antibiotics may make culturing difficult or impossible. In addition, new antibiotics are constantly being introduced, frequently without sufficient study to indicate their true value, their toxicity and the possible complications which might result from their use. Altafur was recently introduced and

enthusiastically promoted as having miraculous qualities; it was proved so toxic that it has been withdrawn from the market. This "salesmanship" approach to therapeutic drugs stands in marked contrast to the extremely careful objective evaluation of the sulfonamide drugs, penicillin and streptomycin in the 1940's when the need for antibiotics was greater but the work was under the control of independent research workers and not the drug companies. It also must be recalled that the contaminated wound usually contains several varieties of bacteria, and when prophylactic antibiotics are utilized, the patient runs the risk of having the most susceptible organisms obliterated while the most drug-resistant organisms, commonly the staphylococci, are left a clear field in which to multiply.

PUBLIC HEALTH FACTORS.

The effect of prophylactic antibiotic therapy on public health cannot be disregarded. Statistics reveal that approximately 75 per cent of all antibiotics produced in this country are used prophylactically and 25 per cent therapeutically. It is now well known that the greater the exposure of bacterial organisms to antibiotics, the greater the number of resistant organisms that will emerge. A study[2] at the University of Illinois Hospital in 1952 showed that when erythromycin was introduced, no staphylococci in the hospital personnel or patients were resistant to it. Within six months, two-thirds of the staphylococci were resistant. In another study Pulaski[5,6] showed that a direct variable relationship exists between the amount of antibiotics used in the hospital and the number of staphylococcal infections. This invites a paradox: to decrease hospital infection rates, decrease the administration of antibiotics!

A larger problem also confronts us: The hospital population often becomes carriers, leaves the hospital and distributes organisms in the community. Epidemics resulting from this type of vector are not unknown. When a ward aide goes home carrying a resistant staphylococcus to her child, who then develops an antibiotic-resistant staphylococcal tracheobronchitis, the ultimate responsibility rests with the hospital

and doctors. It is they who, through indiscriminate use of antibiotics, have created the host of resistant organisms with which the community finds it so difficult to cope.

THE NATURAL HISTORY OF TWO SURGICAL WOUNDS.

A. Surgical Wound When Antibiotic Prophylaxis Is Used.

A wound treated prophylactically with antibiotics may heal uneventfully or may become infected. Even if the wound heals uneventfully the patient may still have a toxic or allergic reaction to the antibiotic, and in any event, he or somebody responsible for him will have to pay from $20 to $50 for the medication. If the wound becomes infected, more antibiotics must be given which, though they may cure the infection, again must be paid for, and again the patient undergoes further danger of toxic and allergic reactions or superinfection necessitating the use of still more antibiotics. This patient also runs a greater risk of having an infection with organisms which develop cross-resistance to most or all antibiotics, making treatment extremely difficult.

B. Surgical Wound Without Antibiotic Prophylaxis.

This wound may heal uneventfully and usually does so no matter what procedure has been utilized; the wound may also become infected. As long as we are able to recognize wound infection by the signs of pain, redness, swelling, fever and leukocytosis, that is to say, as long as we can diagnose infection in this early stage of rapidly dividing organisms, institution of therapeutic dosage of antibiotics when blood supply is intact, usually will result in rapid subsidence of infection without significant tissue necrosis. We have the added advantage of being able to take a culture at the onset of therapy, so that our therapeutic efforts will be based on fact and not on assumption. If a patient is to have surgery and will not be observed for a significant period of time, there might be excuse for the use of antibiotic prophylaxis; but this situation occurs only rarely, and one may inquire why it should occur at all. When the patient is to be seen at regular intervals, the necessity for prophylaxis is seriously questioned. It is

difficult to conceive of an infection so virulent that it could cause serious tissue destruction or systemic spread in the short period of time between doctors' visits and yet not alarm the nursing and house staff with significant objective signs.

THE CASE AGAINST ANTIBIOTIC PROPHYLAXIS.

It seems that under ordinary circumstances the following conclusions are justifiable: *1.* The usefulness of antibiotic prophylaxis has not been proven. *2.* The expense is great, and when multiplied by many hospital admissions, becomes astronomical. *3.* The public health problem is increasing and will continue to do so. *4.* Toxic and allergic reactions are common. *5.* The basic usefulness of antibiotics is being destroyed by indiscriminate prophylactic use.

Several statistical reports have been published in which similar series of cases were compared on the basis of whether or not antibiotic therapy was utilized; the wound infection rates have been equal or frequently higher in the antibiotic group. To my knowledge such a comparison has not been made for major head and neck cases. Since I stopped using prophylactic antibiotic therapy around the end of 1959, I was able to compare the infection rate in my 1959 cases, when practically all patients having major head and neck surgery were given prophylactic antibiotic therapy, with the 1960 cases when no prophylactic antibiotic therapy was used. The material included comparable numbers of such types as laryngectomies, neck dissections, composite operations of various sorts, iliac bone grafts, etc. In 1959, five significant infections occurred, and in 1960, there were three.

If this approach seems radical in that the physician may feel guilty that he is not doing everything possible for the patient, it may, with equal justification, be said that the method is conservative and that by withholding antibiotics until a clear need for them exists he may really be doing all he can for the patient.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1961-1962 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, executive secretary-treasurer of the Academy, 15 Second Street S. W., Rochester, Minnesota. Registrations should be completed before August 15.

CONGENITAL ANOMALIES OF THE ESOPHAGUS IN THE VERY YOUNG INFANT.*†

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Congenital anomalies of the esophagus in the newborn and very young infants are doubly important, because in addition to interfering with nutrition they may result in aspiration and pneumonitis. The latter usually poses a greater threat to early life and since its symptoms are the most dramatic, the esophageal picture may be masked by the respiratory picture.

Lifesaving early diagnosis of these anomalies is difficult even with endoscopy and roentgenology. They must be suspected in all instances of unusual respiratory problems of tiny infants, especially if there is a hint of dysphagia.

Many such patients have come to our Los Angeles Childrens Hospital and they have been used to attempt to set up a differential diagnosis. There is a long list of such conditions, too numerous to describe in detail in a presentation as this, but they will be listed and one series, that of chalasia, will be used to point out an interesting possible answer to one argument of cause and effect. These patients can be divided into two general groups, that is, those who demonstrate a congenital anatomical defect and those who seem to have a neuropathological disturbance.

TRACHEO-ESOPHAGEAL FISTULAS WITH ATRESIA.

Tracheo-esophageal fistulas with atresia of the esophagus make up the greater part of this first anatomical group. This condition is suggested by dysphagia at the time of the new-

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born infant's first attempt at feeding, with immediate coughing, choking and prompt regurgitation of feedings. There usually seems to be excessive mucus or secretions in the mouth and nose. Failure of a soft catheter to pass easily, strongly suggests the diagnosis. X-ray studies with an opaque medium are necessary to confirm it. Treatment is surgical. Postoperatively these atresias have added a new chapter to the book of endoscopic treatment. Cicatricial strictures at the anastomotic site have to be dilated; sometimes sutures have to be removed; recurrent tracheo-esophageal fistulas have to be diagnosed and demonstrated. In some patients perfect anastomosis and healing have resulted in an excellent esophageal lumen, but dysphagia has persisted. This must be due to a neuropathological disturbance of the swallowing mechanism.

H-TYPE TRACHEO-ESOPHAGEAL FISTULAS.

H-type tracheo-esophageal fistulas are much more rare and their diagnosis much more difficult. If undiagnosed, these infants usually die with recurrent aspiration pneumonia. Usually there is exhibited the classical triad of symptoms, that is:

1. Paroxysmal attacks of choking, coughing and cyanosis with feedings, *greatest with liquids.* Placing the patient in a prone position after feeding will sometimes start these symptoms or aggravate them. Often this same patient can take semi-solids without difficulty.

2. Abnormal distention of the abdomen from increased air in the stomach and intestines because of direct communication from the tracheobronchial tree into the esophagus. This is usually greatest and often dramatic when these patients cry or become choked with their feedings.

3. Recurrent aspiration pneumonitis. This constitutes the greatest threat to the patient's life and is the most frequent cause of death if the condition remains undiagnosed.

A fourth finding, sometimes present and very suggestive of this condition, is the passage of very frothy stools.

These patients with H-type tracheo-esophageal fistulas have a lesser degree of increased pharyngeal mucus than do those who have an atresia of the esophagus, and, of course, a soft catheter can be passed. The treatment is surgical, and since most of these fistulas are very high, sometimes just subglottic, the superior mediastinal approach can often be considered first in their repair.

Recurrent postoperative tracheo-esophageal fistulas after repair of a T E F with atresia, are easier to demonstrate radiographically and at endoscopy. In all our instances a blast of air up the scope on expiration was noted with the scope in the esophagus.

CONGENITAL STRICTURES.

Congenital strictures are fairly common anatomical defects. Since fluids usually pass these strictures without difficulty, symptoms and findings of dysphagia do not appear until the little patient is old enough to begin taking semi-solid or solid foods. Increased pharyngeal mucus, dysphagia, regurgitation and aspiration are much less dramatic. Diagnosis is made by esophagram and esophagoscopy. Most congenital strictures can be treated successfully by dilatation.

DIAPHRAGMATIC HERNIAS.

Diaphragmatic hernias of the type in which the diaphragmatic opening is too large is not an anomaly of the esophagus, but it presents the same symptoms of dysphagia, regurgitation and aspiration pneumonitis, and so must be included in this differential diagnosis. In the newborn the opening may be huge and all of the stomach and part of the intestines in the chest. In other patients it may be much less, and difficult to diagnose. Usually a chest X-ray is taken because of the pneumonitis and then the real pathology indicated. The condition is confirmed by X-ray examination with a soft catheter or opaque oil in the esophagus and stomach. The treatment is surgical narrowing of the diaphragmatic hiatus. Esophagogastric and para-esophageal hiatal hernias will be considered later.

CHALASIA.

Chalasia is a condition being seen with increasing frequency in very early life. This cardio-esophageal relaxation is probably a defect of neuromuscular function and is the first of the neuropathological defects to be considered. Another reason for considering it first is that I have a feeling that chalasia was the first underlying causative factor in the production of many conditions that later were diagnosed as congenital short esophagus, thoracic stomach, hiatal hernia and even esophagitis with or without stenosis. We have diagnosed chalasia in the first few months of life. Usually there is a history of difficulty in feeding since birth with choking and vomiting, especially when the child strains or cries. The diagnosis is demonstrated by fluoroscopic examination. With no evidence of obstruction in the esophagus, the reflux of gastric contents through a patulous, wide cardiac orifice is excellently demonstrated with digital pressure over the infant's stomach. This reflux of gastric secretions usually causes some degree of esophagitis which may be ulcerative and bleeding, even to the point of great anemia. The subsequent healing and scarring of this esophagitis must be the cause of many a stenosed and shortened esophagus and these finally result in pulling some of the stomach up through the diaphragmatic hiatus, giving a picture of so-called congenital short esophagus, or hiatal hernia. At esophagoscopy the relaxed or wide open esophagogastric function is easily demonstrated.

CARDIOSPASM.

Cardiospasm, a constant spastic contraction of the cardiac opening of the esophagus, sometimes called achalasia, is frequently demonstrated in the very young infant with dysphagia and regurgitation. The diagnosis is indicated by fluoroscopic and X-ray examination demonstrating smooth obstruction at the cardia with moderate to severe dilatation of the esophagus above this area. Esophagoscopic examination confirms the diagnosis when the scope passes on into the stomach after only short resistance at the cardia. Cardiospasm in the very

young infant seems to respond to dilatation therapy more readily than in the older patients. Surgical correction has not obtained the most desirable end-results as too large an opening has resulted in reflux of gastric contents, ulcerative esophagitis and sometimes great blood losses; at other times this surgery has resulted in cicatrizing stenosis and the need for more dilatations.

Many other, but rarer, neurogenic causes of dysphagia have been encountered in the newborn and very young infant. The glossopharyngeal nerve, the second division of the Vth nerve and the superior laryngeal nerve carry afferent stimuli of the buccal pharyngeal phase of the swallowing mechanism to the swallowing center in the floor of the fourth ventricle. Peristalsis in the esophageal phase is completed by the intrinsic myenteric plexus. The relaxation, the emptying and then closing of the ampullary end of the esophagus in the cardiogastric phase, is the result of action of the vagus, the sympathetic and the parasympathetic plexuses. Disturbances of any of these can result in the dysphagias we are discussing. Cerebral hypoplasia, bulbar paralysis and agenesis of the brain are examples. Prematurity, hypoxia or anoxia due to obstruction of air passages, bruising or edema of the brain may be causes. In these patients there was inability to swallow from birth, with the presence of much mucus in the mouth and pharynx and sometimes with gagging, choking, coughing and regurgitation through the nose. In a few of these patients the symptoms were transient. Neurogenic abnormalities should be suspected when no anatomical abnormality is demonstrated by X-ray or by esophagoscopy. Absent gag reflexes, paralyses and other clinical findings of central nervous system disease may help make or suggest the diagnosis.

A small but interesting group has been those patients demonstrating a flacid cricopharyngeus causing frequent regurgitation and aspiration pneumonitis. Esophagoscopy readily demonstrated a completely relaxed, wide open esophageal introitus.

Patients with amyotonia congenita and the Reilly-Day syn-

drome or familial autonomic dysfunction often develop dysphagia as their disease progresses. Again, repeated aspiration pneumonitis, because of dysphagia and great weakness of the cough reflex, may be their presenting picture and often is the cause of their death. Often tracheo-esophageal fistulas (without atresia) are suspected.

Finally, many single rare instances of dysfunction of the esophagus have been encountered. Impaired or uncoordinated peristaltic activity, spasms of just parts of the esophagus are examples. Non-sphincteric spasm and prolonged evacuation time of the esophagus are indicative.

It will be noted that vasular anomalies have not been included in the foregoing definitive diagnosis of dysphagia. Rarely do such anomalies cause esophageal obstruction or dysfunction but rather result in symptoms of respiratory impairment.

In summary, an attempt has been made to illustrate the many possible anatomic or neurogenic abnormalities that may result in dysphagia of the newborn or tiny infant, causing life threatening aspiration pneumonitis and malnutrition.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The joint annual meeting of the North Carolina Eye, Ear, Nose and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held at the Francis Marion Hotel, Charleston, South Carolina. The dates are September 11-12, 1961. An excellent program has been arranged. There will be three guest ophthalmologists and three otolaryngologists. There will be entertainment features for both the physicians and ladies.

For further information address Roderick Macdonald, M.D., Secretary and Treasurer, 330 East Main St., Rock Hill, S. C.

A TECHNIQUE TO PREVENT BLEEDING FOLLOWING TONSILLECTOMY AND ADENOIDECTOMY.

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Baltimore, Maryland.

This study consists of 773 consecutive tonsillectomies and adenoidectomies without one single case of postoperative bleeding. The definition of no postoperative bleeding as used in this study follows: Immediately following operation approximately 75 per cent of the patients had no vomiting in the recovery room; the other 25 per cent vomited approximately 75 cc of old dark blood and mucus. While in Trendelenburg position there was no blood evident or only a few drops, usually from the nose. The average time in the recovery room was 45 minutes. Upon return to the ward there was very little vomiting and from none to a few cc of blood-tinged mucus evident on the gauze used to wipe the patient's mouth. From that time to complete recovery from the operation there was no bleeding whatsoever.

Proctor[1] gives an excellent definition of bleeding. He describes "slight bleeding as that which requires no specific therapy; moderate bleeding as that requiring prolongation of hospital stay, postnasal packing, or other specific treatment not including blood transfusion or resuturing; and severe bleeding as that which requires blood transfusion or resuturing."

A review of papers dealing with prevention of postoperative bleeding following tonsillectomy and adenoidectomy report slightly under 1 per cent to 20 per cent postoperative hemorrhage. Numerous papers are devoted to detailed studies of the effects of Vitamin C, Vitamin K, Aspirin, Aspergum, local and systemic antibiotics, blood studies including bleeding and clotting time,[2] etc. Many of these reports contradict each other. Others show a definite decrease in the percentage of

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postoperative hemorrhage by a single or non-use, or a combination of the various factors stated above.

The author's experience with 773 tonsillectomies and adenoidectomies without one single case of postoperative bleeding justifies this report. All 773 of these patients were consecutive admissions to the Church Home Hospital, Baltimore, Maryland, between April, 1947 and January, 1961. No operations were done in July, August, and early September because of high temperature and humidity during this period and also because of the possibility of acute poliomyelitis being present. Patients were in the usual ages of from three years to adults. Approximately four-fifths of these patients were children. All patients had tonsillectomies and adenoidectomies based on the usual criteria such as rheumatic fever, arthritis, impaired hearing, tonsillitis, postnasal obstruction, asthma, asthmatic bronchitis, etc.

Following complete medical history and ear, nose, and throat history and examination, those patients who needed tonsillectomies and adenoidectomies were placed on the following dosage for four to six weeks preoperatively:

Each child received one teaspoonful of each preparation daily; adults the equivalent in larger doses. The following ingredients are listed as contained in these easily obtainable preparations which build up the individual's general health. It is impossible to specify precisely which individual ingredient is the effective agent.

1. Paladac by Parke Davis which contains for each 4 cc:

Vitamin B 12 Crystaline	5 mcg.
Vitamin C	50 mg.
Vitamin A	5000 units
Vitamin D	1000 units
Vitamin B1 (thiamine Hcl)	5 mg.
Vitamin B2 (Riboflavin)	3 mg.
Vitamin B6 (Pyridoxine Hcl)	1 mg.
Nicotinamide	20 mg.
Pantothenic Acid (Na. Salt)	5 mg.

2. Rubraton Elixir by Squibb—ea. 5 cc:

Iron (Elemental) as ferr. Ammonium Cit. and Colloidal Iron	(U.S.P.) 38 mg.
B1 (Thiamine Mononitrate)	1 mg.
B2 (Riboflavin)	1 mg.
Vitamin B12—activity concentrate	4.0 mcg.
Vitamin B6—(Pyridoxine Hcl)	9.5 mg.
Vitamin D—(Panthenol)	1.5 mg.
Niacinamide	5.0 mg.

These easy to take liquid preparations are given because many children could not swallow capsules. Through experience with these preparations it is felt that they definitely improve the appetite, increase the hemoglobin, and improve the general health and well being of the patient. It is one's general impression that the frequency of upper respiratory infections is also reduced. The patients were also advised to take a well balanced diet. Parents were advised not to give aspirin unless absolutely necessary.

Before admission to the hospital for tonsillectomies and adenoidectomies, patients were screened in the following manner: In no case was a patient admitted if there was any sign of upper respiratory infection, known exposure to infectious disease, or evidence of any acute disease. No patients were admitted unless the hemoglobin was at least 70 per cent as measured by the Hayden-Hauser colorimeter method, white blood count was under 10,500, and temperature was 98.6 or under by mouth. In the last quarter of this study no patient was admitted unless bleeding time was 180 seconds or less and the clotting time six minutes or less. Not much significance is generally attached to bleeding and clotting time; however, when these tests are done routinely one finds at least 15 per cent with abnormally high tests. Those patients not meeting the above test were placed on Rutin 60 mgms. daily, Duo C.V.P. (Citrus Flavinoid Compound), and several courses of Hykinone Vitamin K, 5 mg. They were then rechecked and were usually ready for tonsillectomies and adenoidectomies within four to six weeks.

Upon admission to the hospital routine hospital physical

examination and blood and urine studies were done. All patients without exception (unless sensitive to penicillin—and in these cases another antibiotic was given) received 300,000 units of penicillin intramuscularly, 200 to 500 mg. ascorbic acid by mouth, and 5 mg. Hykinone Vitamin K hypodermically. These dosages were repeated that evening. All patients were again screened the next morning, and operation was cancelled if they did not meet the above requirements. The above mentioned Hykinone Vitamin K and ascorbic acid are routinely given to supply any possible deficiency of these vitamins. Penicillin (or other antibiotic) is given to protect against the possibility of infection and also to retard secondary infection postoperatively in the tonsillar fossae. Atropine was not used in children unless 12 or more years of age. Atropine was never used in this age group because a hypodermic just before operation causes excitement; atropine counteracts the effect of postoperative sedation; and those children not excited have very little secretion from the nasopharynx, larynx, and trachea; any secretions present are thin. Atropine tends to make secretions thick and thus provides more chance for atelectasis. There were no cases of atelectasis in this series. Pentobarbital (or equivalent) suppositories 30, 60, or 90 mg. are given, according to the weight of the individual. Anesthesia was Nitrous Oxide, oxygen, and drip and insufflation ether. Adults received Morphia 10 mg., Atropine 0.4 mg. and were anesthetized with pentothal, nitrous oxide, curare, and all were intubated.

When using pentothal for tonsillectomy the patient was intubated to protect against laryngeal spasm and the associated respiratory difficulty. All cases were done at 8 a.m. and anesthesia in all cases was kept as light as possible. The patient was in the Trendelenburg position with suction available at all times. Operative procedure was a modified Crowe technique, using the Davis-Crowe mouth gag. Adenoids were removed with adenoid curette, and after wiping the nasopharynx with Crowe fluffs several times, a large sponge was inserted into the nasopharynx while the first tonsil was being removed. This was replaced by a standard adenoid plug before starting the second tonsil. A snare was never used.

The scissors blunt dissection technique was used throughout. After the tonsil is removed a rolled sponge on a Kelly clamp is firmly held in the fossa for about 20 seconds. This is repeated three to four times. A Kelly clamp is then used to grasp tissue between the anterior and posterior pillar at the very end of the lower pole of the tonsillar fossae. Using Martin cutting needles with third lengths 00 plain catgut tied on, the needle is inserted deep through *both pillars* at the end of the lower pole, while the tissue in between is elevated with a Kelly clamp mentioned above. One should palpate the area for the carotid artery and avoid suturing deep enough to penetrate this vessel. After completing a figure eight mattress suture the Kelly is released and the sutures tied. This almost always stops all bleeding in the fossa and adjoining mucous membranes and brings together and covers the usually opened area where the tonsil meets the base of the tongue. It should be noted that this technique is entirely different from Emerson's[3]; he sutures the entire fossa. If any other bleeding point is noted this should be immediately tied off. In this series, an average of two sutures were used for each case. After both tonsils have been removed the mouth gag should be lowered for 30 seconds or more to relax the tissues and upon re-opening the gag, recheck for bleeding.

Postoperatively, in the recovery room, all patients immediately received 300,000 units of penicillin intramuscularly and 5 mgs. Hykinone Vitamin K hypodermically. By retarding infection and speeding healing the chance of bleeding is greatly diminished. This is repeated at 8 p.m. and also the next morning.

The head of the bed is elevated after consciousness, ice collar is used constantly, patient is placed on liquid diet and forced fluids. No medication for pain is given except elixir of pyribenzamine, if necessary, for children; morphine, if necessary, for adults. Patients are discharged the following morning and should remain in bed, except for bathroom privileges, for four to six days. No patient was allowed aspirin in any form. A soft diet for one week consisted of

anything the patient liked, in soft form. High sugar intake was stressed, particularly the first few days, because this seems to make them feel better. The patient was advised to notify the surgeon if the throat was sore. Surprisingly, this was a *rare occurrence* (about 5 per cent). If called, treatment advised was elixir of pyribenzamine 1-6 teaspoonfuls per day for pain. This is given because of its local anesthetic action, reduction of edema, and its ability to counteract trauma following the surgical procedure. In almost all instances the sore throat was relieved immediately with the first dose. If there was evidence of infection around a suture, with temperature of 102. or more, oral penicillin 200,000 units t.i.d. was given for four or five days. The preoperative vitamin therapy was started postoperatively as soon as possible.

All patients received the following gargle which consisted of one-fourth teaspoon of salt and one-fourth teaspoon of sodium bicarbonate in warm water, at least four glasses daily. After the fourth or fifth day in bed, it was advised that the patient get up slowly and do more and more each day. In most cases children were kept home from school for a period of approximately two weeks. The patient was seen in the office for examination at the end of the two-week period. In all cases, the fossae had healed very nicely. If the patient had responded rapidly he was allowed to go to school sooner, but only after a checkup by the surgeon. All patients were again seen two months after the two-week visit.

In summary, a rigidly followed technique has been developed to prevent bleeding following tonsillectomy. The patient must be evaluated from his medical and ear, nose, and throat history and examination, and he must be given help in recovering from whatever disease has contributed to his ear, nose, and throat complaint. This is done with improved diet, minerals, and vitamins, and a period of time before the actual operation. This is usually from four to six weeks. Before entering the hospital he must have a hemoglobin of at least 70 per cent, white blood count under 11,000, and normal temperature. He must also have normal bleeding and clotting time and be free of acute infection. Once in the hospital he is

given penicillin and Vitamins C and K to protect against infection and possible deficiency of Vitamins C and K. This combination would appear to be an important factor in preventing the bleeding which could occur in adenoid tissue or mucous membranes. By use of the figure eight mattress sutures to restore the anatomy by approximating mucous membranes at the lower pole and base of the tongue and to stop arterial and venous bleeding, much of the problem is solved. With antibiotics and vitamins postoperatively, the secondary bleeding associated with infection is controlled.

Avoidance of aspirin and substitution of elixir of pyribenzamine prevents any abnormality in the clotting mechanism attributed to aspirin.

Simple postoperative saline and soda gargles, plus adequate rest, help rapid recovery of the tissues. Postoperative maintenance of nutrition with diet and vitamin therapy further help to restore the tissues to normal. It is difficult to determine exactly which portion of this technique is responsible for the results. It is my feeling that it is a combination of all of the factors discussed that account for the success of this technique.

In conclusion, the object of this report is twofold: *1.* to draw attention to the great significance of minor detail and strict adherence to simple procedures and therapy which apparently can prevent hemorrhage following tonsillectomy and adenoidectomy; *2.* to hope that other otolaryngologists will try these simple procedures to test further the validity of this report, so that this always annoying and sometimes quite serious complication of tonsillectomy and adenoidectomy can be abolished.

The author thanks Dr. John Bordley, Professor of Otolaryngology at Johns Hopkins Hospital, for his helpful suggestions and his review of this paper.

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PALATAL MYOCLONUS: A REPORT OF TWO CASES.*

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Palatal myoclonus is a relatively rare condition in which the palatal muscles undergo continuous rhythmic contractions. The clonus is most commonly bilateral and often may produce a clicking sound in the ear of the patient. The sound may be heard by the examiner. The rate of the contractions may vary from 10 to 240 per minute. "Muscular tinnitus" is a term applied to the clicking sound, which resembles that made by the snapping together of two fingernails. It has not been established whether the sound is produced by the Eustachian tube snapping open or snapping shut; however, from our observations (described in the first case report) we believe the sound is produced by the tube snapping shut.

The exact cause of palatal myoclonus is unknown. It has been associated with vascular lesions, multiple sclerosis, aneurysm of the vertebral artery, tumor, and various other lesions involving the brain stem or cerebellum.[3,4,7,13] There are several reports of patients who could voluntarily produce the palatal myoclonus[1,6,14] and also of patients who were relieved of the myoclonus by posthypnotic suggestion.[1,15]

In 1953 Erickson and Ablin reviewed the literature and found reference to 134 cases; they added two cases of their own. A review of the literature[1-3,5,7-12,14,15] subsequent to their paper produced an additional 34 cases. With the two cases reported herein, this makes a total of 170 cases.

REPORT OF CASES.

Case 1. A 24-year-old white single woman came to the clinic complaining of intermittent pains between her throat and her ears and a clicking

*From Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

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noise in her ears. The pain and clicking had been present intermittently on the left for two years and on the right for one year.

Her past history was unremarkable except for left simple mastoidectomy at age six, tonsillectomy and adenoidectomy at age eight, and Asian "flu" two years ago; the history revealed no symptoms of brain-stem disease. Her general health had been good. Results of neurologic examination, roentgenologic study of the skull, serologic examination, urinalysis, and determination of hemoglobin, differential blood count and blood sugar were normal. The family history was noncontributory.

Physical examination revealed a tall, thin young woman who appeared to be in good health. Hearing was normal. The tympanic membranes showed no spontaneous rhythmic movement indicative of clonus of the tensor tympani muscle, although with a stethoscope or auditory tube a rhythmic "click" could be heard in both ear canals.

The muscles of the soft palate and Eustachian tube were found to be undergoing rhythmic contractions bilaterally. The movement of the palate could easily be seen either with a nasopharyngeal mirror or when the palate was viewed directly, through the nose, after the nasal membranes were shrunk.

The click in the ears was synchronous with the falling phase of the palatal clonus and was interrupted by swallowing or talking.

We observed the patient for several days, and she seemed to enjoy the attention. She stated that the location of the clicking varied, being now in one ear and now in the other or at times in both ears. The palatal movement, however, was bilateral at all times.

Cocainization of the nasopharynx had no effect on the palatal movement and clicking. Electromyography with an electrode placed in the right Eustachian tube recorded motor-unit activity in bursts lasting from ½ to 1½ seconds and separated by quiet intervals of ½ to 2 seconds. The click, heard by placing a stethoscope over the external canal, occurred about one second after the onset of electrical activity. Regions of the pharynx and external ear, other than the Eustachian orifice, showed no significant electrical activity.

A small rubber balloon at the tip of a cannula was placed within the right Eustachian tube, and the system was connected to a water manometer. Three different examiners observed that the click heard at the external ear canal, the falling phase of the palate, and a rise in the reading of the water manometer all occurred at the same time.

After several days of observation we noted the manifestation of neurotic tendencies in this patient, and we considered strongly the possibility of a hysterical basis for the disorder. She refused to let us attempt hypnosis.

Case 2.—A 42-year-old white man complained of having heard a clicking in his ears for the past two years. The clicking was present much of the time and occurred more often on the left side. The sound had been heard by his wife. Twice yearly for the past 20 years he had had grand mal seizures, for which he took diphenylhydantoin sodium (dilantin sodium).

On examination, his blood pressure was 105 mm. of mercury systolic and 66 mm. diastolic, and the heart rate was 46 per minute; an electroencephalogram and an electrocardiogram were normal. His hearing showed a mild loss of high tone. He had normal tympanic membranes. An audible click could be heard by the examiner with an auditory tube in the left ear of the patient. With each click the palatal musculature, especially the posterior pillar, could be seen to move. The palate moved

at the time of the click. This movement was rhythmic but slightly irregular. The patient was not greatly disturbed by the clicking sound and was, therefore, dismissed after his neurologic examination disclosed that no organic lesion was present.

COMMENT.

Two cases of palatal myoclonus are described. We were able to study the function of the Eustachian tube in Case 1. From our observation of this one case we believe the following to be true: *1.* The clicking noise heard in palatal myoclonus is the result of the walls of the Eustachian tube snapping together. *2.* The palatal muscles that act upon the Eustachian tube open the tube when they contract and allow the tube to close when they relax.

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EIGHTH ARGENTINE CONGRESS OF BRONCHOESOPHAGOLOGY.

The Eighth Argentine Congress of Bronchoesophagology will be dedicated to the memory of the late Chevalier L. Jackson, M.D., of Philadelphia, Penna. This Congress will be held in Tandil, province of Buenos Aires, Argentina, November 17-20, 1961, under the presidency of Dr. Juan Carlos Arauz, assisted by several foreign physicians. For further information write Dr. Juan Blank, Secretary Cangallo 2150 BsAs, Buenos Aires, Argentina, South America.

SEVENTH INTERNATIONAL CONGRESS OF NEUROLOGY.

(Rome, 10-15 September 1961)

FIFTH INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY.

(Rome, 7-13 September 1961).

The Seventh International Congress of Neurology will be held in Rome, September 10-15, 1961, under the auspices of the World Neurologic Federation and of the National Institute for Nervous Diseases and Blindness of Bethesda.

Together with the general Assembly of the International League against Epilepsy and the Vth International Congress of Electroencephalography the first day will be dedicated to the opening Session.

September 11th and 12th will be given respectively to the discussion of Topics I and II, while Topic III and the communications will be represented respectively on the 14th and 15th. Symposia will be held on September 13, a day free of other Congress engagements, to permit an interval between the two parts of the works.

Registration fees and other modalities are the same as those established for the Congress of Neurology. The official banquet and dance will be held in conjunction with this Congress.

Any information is obtainable from the President of the Congress, Prof. Mario Gozzano, or from the Secretary General, Dr. Raffaello Vizioli, Viale Universita 30, Roma.

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(Secretaries of the various societies are requested to keep this information up to date).

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BY

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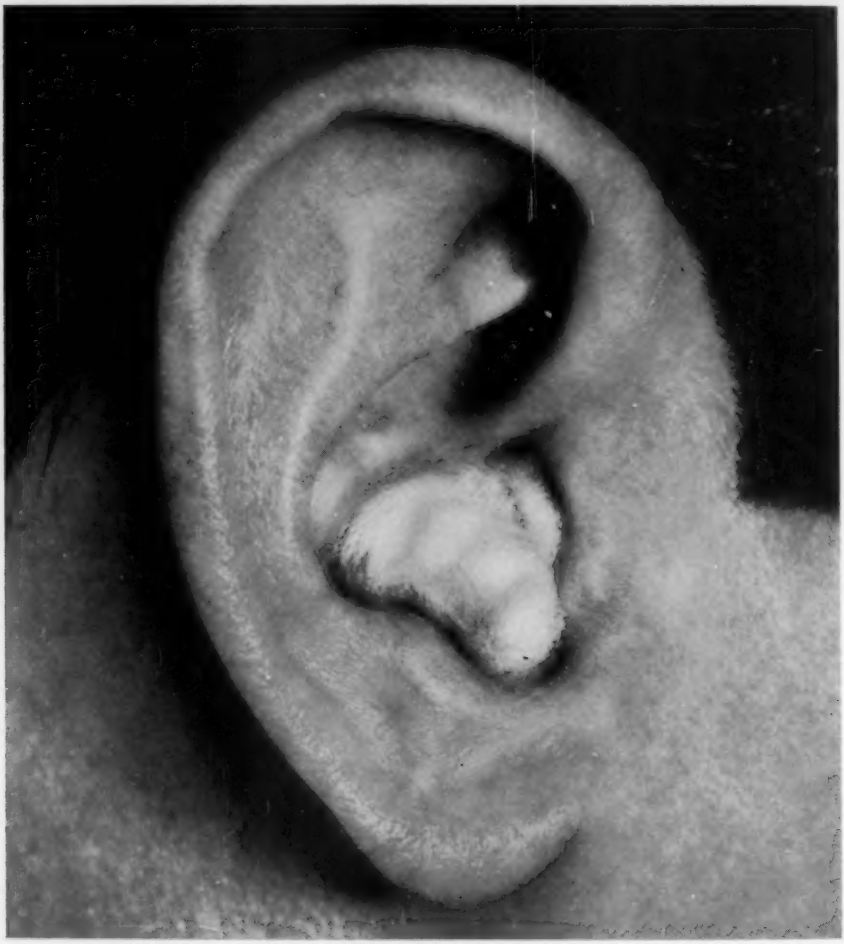

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1. Adams, J.N., Iodo-Niacin in treatment of sinusitis, bronchitis and otitis, *Southwestern Med.* 40:120, 1959.
2. Feinblatt, T.M., Feinblatt, H.M., and Ferguson, E.A., Jr., Treatment of arteriosclerosis and vague abdominal distress with niacinamide hydroiodide (without side-effects), *Am. J. Digest. Dis.* 22:5, 1955.

*U.S. Patent Pending

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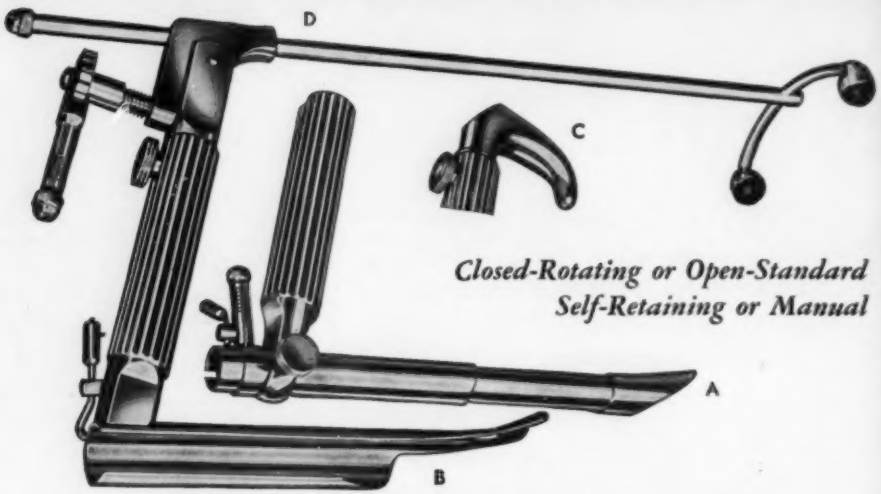

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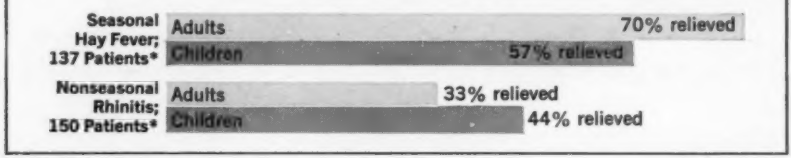

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1. Feinberg, S. M., Feinberg, A. R., and Fisherman, E. W.: J. Indiana M. A. 52:2137 (Dec.) 1959.
*Adapted from authors' table.

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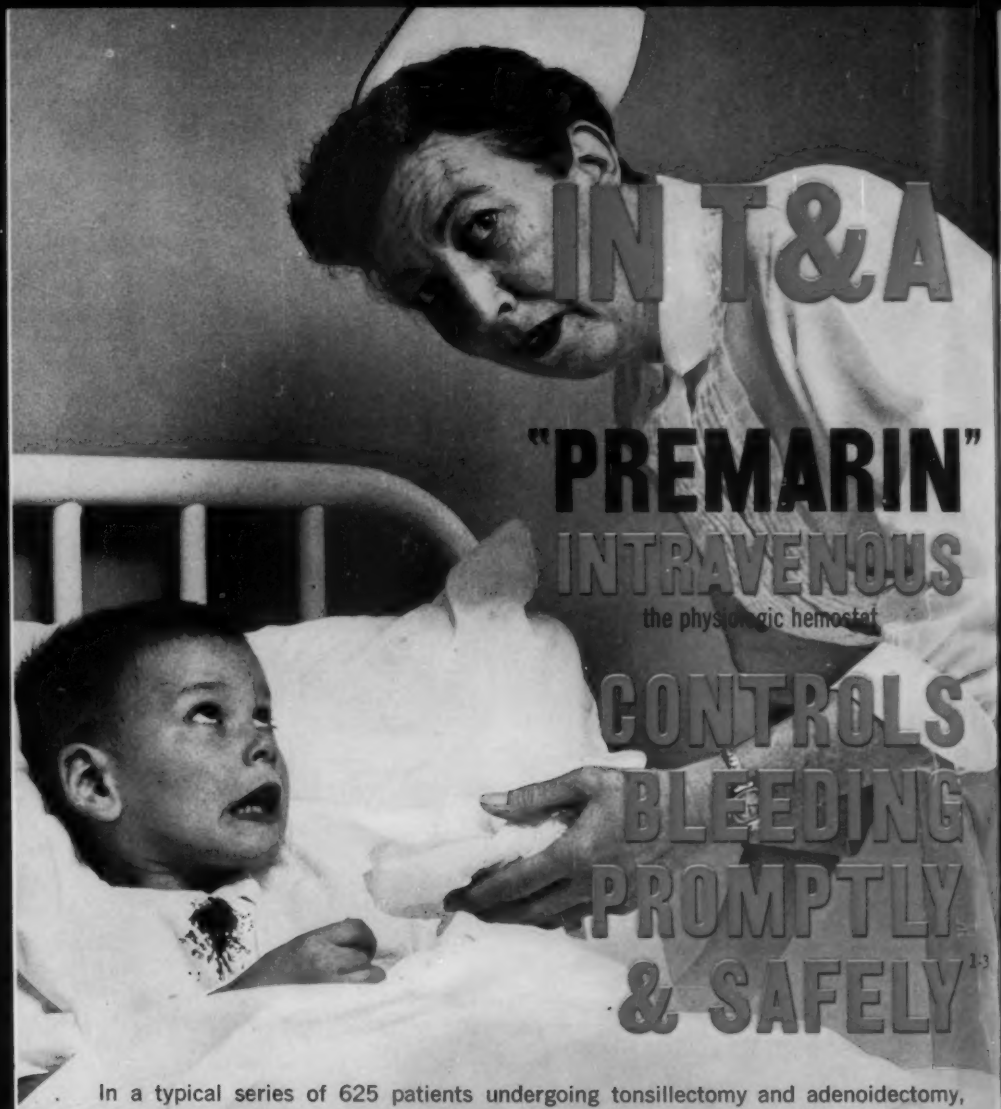

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1. Johnson, J. F.: Paper presented at Symposium on Blood, Wayne State University, Detroit, Michigan, Jan. 18, 1957, cited in M. Science 1:33 (Mar. 25) 1957; Proc. Soc. Exper. Biol. & Med. 94:92 (Jan.) 1957. 2. Servoss, H. M., and Shapiro, F.: Digest Ophth. & Otolaryng. 20:10 (Nov.) 1957. 3. Published and unpublished case reports, Ayerst Laboratories.

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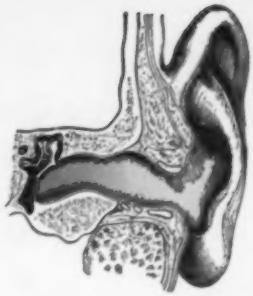

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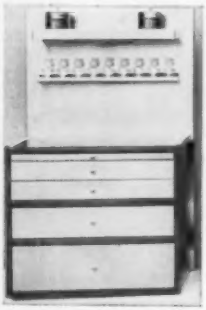

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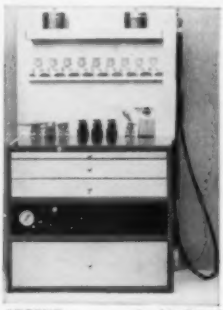

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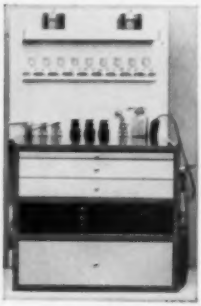

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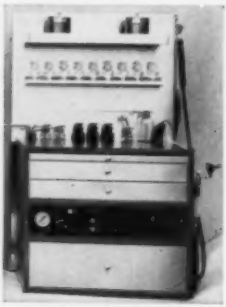

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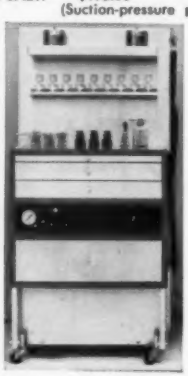

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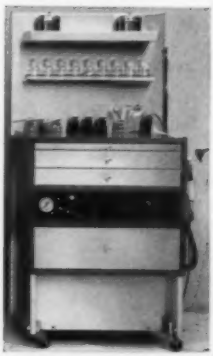

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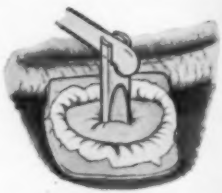

Used when the lenticular process is missing or deficient. Sterile packed in sealed polyethelene packets, containing one 4½ mm length strut on stainless steel ring. No. 1212G

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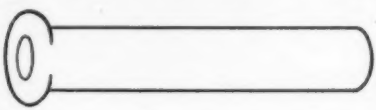

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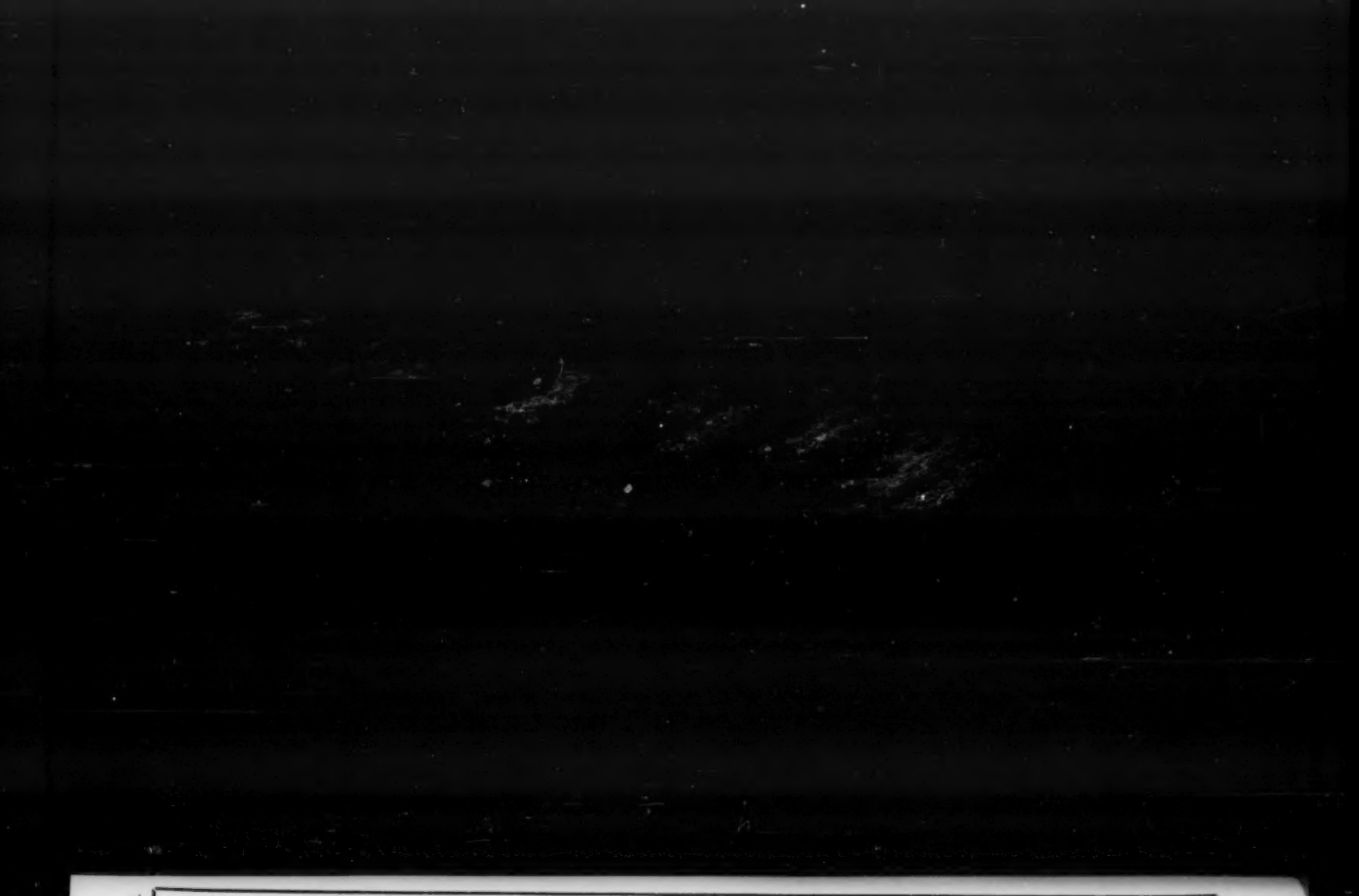

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